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Enviro Control, Inc, Rockville, MD

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16. ABSTRACT

This report documents the results of four case studies of correlations between industrial activity and community disease: (1) copper smelting and respiratory/digestive diseases; (2) steel manufacturing and digestive neoplasms; (3) bituminous coal mining and cardiovascular/respiratory diseases; (4) viscose rayon and cardiovascular diseases. The four correlations were found by statistical analyses correlating county-by-county mortality data with industrial data. The case studies consisted of field investigations at specific industrial sites to explore the plausibility of causal relationships between the selected industries and the associated diseases. Overall purpose of the work is to assist in the development of objective and systematic methodology for identifying industrial origins of community disease. Results of the case studies provide a basis for assessing the feasibility of finding causal industry-disease associations by correlating county-by-county industry and mortality data.

In three out of the four case studies, evidence was found of possible relationships between the industries and the correlated diseases. This evidence varied in strength and completeness and in no case was sufficient to enable definite conclusions to be drawn regarding the existence of cause-and-effect relationships. However, as far as they go, the results indicate that the multiple regression analysis of county data may be a useful technique for identifying certain types of industry-disease relationships.

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INVESTIGATION OF SELECTED CORRELATIONS BETWEEN INDUSTRIAL ACTIVITY AND COMMUNITY DISEASE

Contract No. 68-01-4304

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NOTICE

This report has been reviewed by the Office of Toxic Substances, Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Environmental Protection Agency. Mention of trade names or commercial products is for purposes of clarity only and does not constitute endorsement or recommendation for use.

PREFACE

This investigation was conducted by Enviro Control, Inc., under contract with the U.S. Environmental Protection Agency's Office of Toxic Substances (Contract No. EPA 68-01-4304).

Each chapter of this report was the responsibility of one of the six principal authors and, for the most part, prepared by that author, with contributions as needed from the others. The chapters and their principal authors are as follows:

	Chapter	Principal Author
I	Introduction	Willard W. Perry
II	Summary	
III	Conclusions and Recommendations	
IV	Methodology	Robert Goldsmith
V	Copper Smelting Case Study	Margaret E. Mattson
VI	Steel Manufacturing Case Study	Mary A. Urbanek
VII	Bituminous Coal Mining Case Study	Carl B. Bailey
VIII	Viscose Rayon Case Study	Bertram W. Berney

The authors wish to acknowledge the many technical and editorial contributions of John D. Morton who served as Program Manager throughout the study. Although not listed as one of the principal authors, Dr. Tee L. Guidotti contributed several important pieces on the etiology of the diseases of interest. These research papers are included in the appendices under his name.

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Chapter I

INTRODUCTION

A. *STUDY PURPOSE AND OBJECTIVES*

This report documents the methods and results of an investigation of selected correlations between industrial activity and community disease. It constitutes the second phase of an EPA-sponsored effort to develop systematic and objective methodology for investigating the possible industrial origins of chronic community disease. The first phase, performed by System Sciences, Inc. (SSI), was a multiple regression analysis of county mortality and industry data to identify statistical correlations between specific industries and diseases [1]. The purpose of the present study is to investigate these statistically derived industry-disease correlations to determine if they have a plausible physical and etiological basis. By so doing, the study provides a measure of the effectiveness of the multiple regression analysis for generating valid associations between industry and community disease.

In this light, the ultimate objective of the study is methodological, i.e., to develop and evaluate methods for analyzing and investigating regression-derived industry-disease correlations to assess their physical and etiological plausibility. These methods vary in complexity from simple screening analyses and literature surveys to in-depth epidemiological field investigations. To provide the basis for methodology development, the specific objectives of the study are:

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[1] System Sciences, Inc., *Investigation Into the Industrial Correlates of Environment Related Mortality*, 1976.

(1) To screen the regression-derived correlations to eliminate those that are weak and the likely result of confounding factors, and to identify those that are statistically sound and promising candidates for true associations.

(2) To perform field studies of a selected number of industry-disease correlations to investigate the possible existence of causal relationships by the verification (or lack of verification) of: (a) an identifiable toxic substance emitted by the industry, (b) a feasible pathway by which the toxic substance can reach a population at risk, (c) the presence of the toxic agent in the environment to which the population at risk is exposed, (d) elevated mortality or morbidity in the population at risk, and (e) a known etiological relationship between the disease and the toxic agent.

(3) To review the results of (1) and (2) above and evaluate the effectiveness of the multiple regression approach for identifying possible industrial sources of community disease.

Most of the study effort was devoted to the field investigations which were conducted over a 12-month period. The objective of these investigations was not to prove or disprove that the industry was the cause of the associated disease. Rather, it was to determine if a rational causal hypothesis could be derived from and supported by readily available field data. The degree of "proof" that can be associated with these hypotheses depends upon the amount and quality of field data and the evidence they provide, which is generally quite limited. Thus, the field investigations are a first step in the more extensive effort required to establish convincing proof of the existence of true relationships between the industries and the associated diseases. They are, in reality, investigations to assess the plausibility of cause-and-effect associations identified by other means. Their purpose is to provide the basis for an intermediate decision, before the commitment of the much more extensive effort required to establish proof.

B. STUDY APPROACH

To satisfy the study objectives, in-depth case studies of four industry-disease correlations were conducted. Three of these correlations were selected by us from those generated by the SSI multiple regression analysis after we had made a thorough screening and validation of the regression results. The fourth correlation was a subjective correlation arising from community complaints and from an independent examination of county mortality data. The fourth correlation was included in the investigations to test an alternative method of selection and to broaden the chance of useful field studies if the regression method failed.

The four industry-disease correlations for which in-depth field investigations were conducted were:

- Copper Smelting -Digestive Diseases
- Steel Manufacturing -Cancer of the Digestive Organs
- Bituminous Coal Mining -Respiratory and Cardiovascular Diseases
- Viscose Rayon Manufacturing -Cardiovascular Diseases

The field investigations consisted of case studies of specific sites, each containing a suspect industry and a population at risk. To determine if disease-specific mortality was greater than expected in the population at risk, comparisons were made with demographically similar communities not containing the industry. Where mortality was elevated in the population at risk, data collected on industrial processes, emissions and effluents, air and water quality, meteorology and hydrology, demographics, etiology, and any other pertinent factors provided the basis for interpreting the apparent industry-disease association in terms of possible causal hypotheses. When available, morbidity data were collected and analyzed. For each of the four case studies, at least two separate sites were investigated to provide a measure of replication consistency. In general, the first site was used for development of a set of causal hypotheses that were then tested for applicability at a second site.

Due to the wide variation in the location of the study sites and in the type and amount of data available in the different states and regions, the case studies differed considerably in the specific methods and analyses used. This turned out to be particularly useful in the development of methodology, since it offered the study team the opportunity to experiment with and evaluate a variety of techniques. In general though, the investigation of whether or not the population at risk experienced higher-than-expected mortality was accomplished through comparisons of age-adjusted, disease-specific mortality rates with those of demographically similar (matched-control) areas.

The study was conducted over a 16-month period, in which the first four months' effort consisted of the selection of industry-disease correlations to be investigated and the detailed planning of the case studies. The results of this initial planning phase have been documented earlier [2]. The four case studies, documented in the present report, were carried out over the 12-month period following the initial planning phase.

C. *BACKGROUND*

For the most part, EPA's motivation for investigating potential environmental health problems and sources has been laboratory and occupational health findings or catastrophic pollution incidents which indicate that a particular substance is harmful. Since there are a great many potential problems and a limited budget for investigations, EPA can pursue only a small fraction of the possible environmental health problems that are posed by the numerous researchers and interest groups. In many instances, it is the enthusiasm and persistence of the proponents of the investigation which determine the priority of a proposed study.

In the search for more objective methods for identifying critical environmental health problems that should be studied, EPA initiated the methodological development and evaluation effort of which this study is a part. Objectivity is achieved by relying on statistical correlations to indicate the industrial sources that are potentially the most hazardous to community health. The correlations are made between the level of industrial activity for U.S. counties and the county mortality rates, for various industries and diseases. By this objective means, the particular industries that are most strongly correlated with specific diseases are identified. These correlations are then investigated epidemiologically, to determine if a reasonable causal hypothesis can be developed to explain them. In the development of this basic methodology, SSI performed the statistical work to establish the industry-disease correlations, and this present study describes the work by Enviro Control, Inc. (ECI) to investigate the causal basis for the correlations.

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[2] Enviro Control, Inc., *Plan for the Investigation of Selected Correlations Between Industrial Activity and Community Disease*, Volumes I and II, February 1977.

It has been recognized that the success of the regression methodology is not a foregone conclusion. There are a number of reasons why a multiple regression analysis of county industry and disease data may not be very effective in identifying industrial sources of community diseases. First, the industry categories are quite broad and contain many different kinds of industry. Thus, a particular industry that is strongly contaminating the community may represent only a small part of the industrial category, and therefore show up as a weak correlation. Second, the emissions of harmful substances could be largely a plant management problem which occurs in a small portion of the plants comprising a given industry, again resulting in a weak correlation. Third, the measure of industrial activity for a county may be a poor measure of the amount of disease-causing agents emitted by the industry. Finally, the polluting industrial facility in a county may affect only a small portion of the county population or it may affect the populations in adjacent counties, and the increase in the overall mortality rate for a county with the industry may be too small to detect.

For these reasons, it was by no means clear that the regression analysis would yield valid correlations. True industry-disease effects could be lost in the noise, and any correlations that were found could be the result of other unknown factors. Consequently, the principal purpose of the investigation reported here is to determine if the correlations that were found have a plausible causal basis. In this way, this study is an evaluation of the feasibility of generating meaningful correlations by means of a multiple regression analysis of county mortality and industry data.

Although the total methodology consisting of the county data correlation analysis together with the epidemiological investigation is unique, a number of studies have been conducted which are relevant to this approach—either to the correlation analysis or to the epidemiological investigation—which lends credence to the basic concept.

Studies based on data from Cancer by County [3] are examples of the correlation analysis approach. The methodology used in these types of studies is exemplified by the publication of Blot et al., entitled "Cancer Mortality in U.S. Counties with Petroleum Industries" (*Science 198*:51-53, 1977). Counties with employment in the petroleum industry over a certain level were selected and compared with a group of "control" counties of comparable demography and geography. Ratios of age-adjusted mortality rates were then compared for 23 cancer sites. This methodology does not consider occupation, location or duration of residence, or lifestyle (e.g., diet and smoking) of the decedents and, therefore, is more useful as a screening tool.

The type of study which we have denoted as a "epidemiological investigation" attempts to clarify an association by considering smaller geographic units, location of residences with respect to the industrial pollution pathway, and specific compounds in the industrial emissions or effluents. An example of this approach is a recent study by Matanoski et al. of cancer mortality near an arsenical pesticide plant in Baltimore, Maryland (NTIS, PB-255-019, May 1976). In this case, census tracts near the plant were compared with tracts at some distance from the plant relative to cancer mortality. Death certificates were obtained, and decedents were categorized by place of last residence. Age-adjusted mortality rates were computed and compared for statistical significance.

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[3] Mason, T. J., and F. W. McKay, *U.S. Cancer Mortality by County: 1950-1969*, DHEW Publ. No. (NIH) 75-780, U.S. Government Printing Office, Washington, D.C. 1973.

D. *OUTLINE OF REMAINDER OF REPORT*

The balance of the report is organized into seven chapters as follows.

Chapter II (Summary)—This is a complete summary of the study consisting of a description of each of the four case studies and a discussion of the results. In the latter, the results of all four case studies are discussed in the light of the study objectives. This constitutes the only integrated discussion of the results of the four cases in this report.

Chapter III (Conclusions and Recommendations)—This presents and discusses the overall conclusions and recommendations derived from the study.

Chapter IV (Study Methodology)—This chapter describes the case study methodology. Also included is a description of the methodology for screening, validating, and selecting the industry-disease correlations for field study.

Chapters V, VI, VII, and VIII (Case Studies)—These chapters provide detailed documentation of the four case studies. Each chapter constitutes a separate case study, presented in the order of: Copper Smelting, Steel Manufacturing, Bituminous Coal Mining, and Viscose Rayon Manufacturing.

Chapter II

SUMMARY

A. *SUMMARY OF CASE STUDIES AND FINDINGS*

1. *Case Study Selection*

To select industry-disease correlations for field investigation, the results of the regression analysis were first subjected to a fairly rigorous screening and validation process. Weak correlations and correlations unfit for study were eliminated from consideration. A progressive series of statistical tests were performed on the remaining correlations to identify those that could be validated by independent statistical means. The final test was a matched-control test in which the mortality rates for the top ten counties in industrial activity were compared to the mortality rates for a set of control counties which were demographically similar to the top ten. For comparison purposes, industry-disease associations were identified by means other than the regression analysis (e.g., occupational associations) and subjected to the same type of validation testing. The correlations that survived these screening and validation procedures are listed in Table II-1.

Four industry-disease correlations were selected from this list for in-depth field investigation. These are listed below.

Industry	Disease	Source of Initial Correlation	Chapter
Copper Smelting	Digestive Disease	Multiple Regression Analysis	V
Steel Manufacturing	Cancer of Digestive Organs	Multiple Regression Analysis	VI
Bituminous Coal Mining	Cardiovascular Disease, Respiratory Disease	Multiple Regression Analysis	VII
Viscose Rayon	Cardiovascular Disease	Other	VIII

Each of these case studies is described and discussed in detail in a separate chapter of this report as noted. This present section gives a brief summary of each investigation and its findings.

Table II-1

CANDIDATE INDUSTRY-DISEASE ASSOCIATIONS FOR FIELD STUDY

SIC	INDUSTRY	DISEASE
3357	Non-Ferrous Wire Drawing	Neoplasm, digestive organs Neoplasm, intestine
102R	Copper Ore Mining*	Acute myocardial infarction
121R	Bituminous Coal Mining*	Respiratory diseases Acute interstitial & bronchopneumonia Bronchitis, emphysema & asthma Emphysema Asthma Other respiratory diseases Major cardiovascular disease Ischemic heart disease Acute myocardial infarction Chronic ischemic heart disease, angina pectoris Diseases of veins, lymphatics & circulatory system
3312	Blast Furnaces*	Neoplasm, digestive organs
2321	Men's Shirts & Nightwear	Major cardiovascular disease Ischemic heart disease
3331	Primary Copper*	Alcoholic cirrhosis of liver Digestive diseases
3421	Cutlery	Respiratory disease
34RR	Fabricated Metal Products	Hypertensive disease
2879	Pesticides & Agricultural Chemicals	Respiratory diseases
2823	Viscose Rayon*	Cardiovascular Disease

*Associations selected for field investigation.

2. *Copper Smelting Case Study*

a. *Basis for Correlation*

The multiple regression analysis showed a relatively strong correlation between the copper mining industry and respiratory, cardiovascular, and digestive diseases in white females. Upon examination and validation of the correlation data, it was found that the copper smelting industry is generally located in the same counties as the mines and is the more logical source of the associated diseases. The matched-control county test supported this view when it resulted in stronger correlations for copper smelting than for copper mining. The strongest of the disease correlations with copper smelting was digestive disease.

b. *Study Site Selection*

For the purpose of causal hypothesis development, three sites were selected in the Arizona counties of Gila and Pinal. These are shown in Table II-2. Note that one of the sites (Hayden) contained two copper smelters; hence, the initial field study involved the investigation of four copper smelters. The criteria for selection were high industrial activity, elevated mortality rates in digestive diseases, absence of other confounding industries, and the ready availability of industry data, air and water quality data, and mortality data. Particular features of the mortality data in Arizona were the inclusion of length of residence information on the death certificates and the availability of most of the death certificate information on computer tape.

Anaconda, Montana, was selected as the primary hypothesis test site. Of all the candidate copper smelting sites, it possessed the largest population at risk for which acceptable mortality data were available,* a characteristic that was felt to be very important since the investigation of the Arizona sites had shown that larger exposed populations were necessary. Also, there were several populations in the vicinity of Anaconda that appeared to be demographically similar to Anaconda's and hence could serve as control populations.

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* Tacoma, Washington, had a larger population but mortality data were not available for the level of subdivisions needed, i.e., for census tracts.

Table II-2. COPPER SMELTING SITES INVESTIGATED

	SITE	COUNTY/STATE	SMELTING COMPANY
HYPOTHESIS DEVELOPMENT	Miami	Gila/Arizona	Inspiration Consolidated Copper Company
	Hayden	Gila/Arizona	Kennecott Copper Corp.; ASARCO
	San Manuel	Pinal/Arizona	Magma Copper Company
HYPOTHESIS TEST	Anaconda	Deer Lodge/Montana	Anaconda Company
	Magna	Salt Lake/Utah	Kennecott Copper Corp.
	Douglas	Cochise/Arizona	Phelps-Dodge Corporation
	Ajo	Pima/Arizona	Phelps-Dodge Corporation
	Morenci	Greenlee/Arizona	Phelps-Dodge Corporation

In the investigation of the Gila and Pinal County sites, mortality, demographic, health, and air quality data were acquired for the entire state. Consequently, it was possible to investigate the three other copper smelting sites in Arizona with little extra effort. These were the Phelps-Dodge plants at Ajo, Morenci, and Douglas. In addition, the data collected in Utah for the steel manufacturing case study permitted the ready investigation of the copper smelting site at Magna, Utah. All told, a total of eight copper smelter sites (nine copper smelters) were investigated: three for hypothesis development and five for hypothesis test. These are listed in Table II-2. This large number of sites was a feature of the copper smelting investigation that was not repeated for any of the other case studies (each of the other three case studies investigated two sites only).

c. *Industry Characterization*

Detailed descriptions of both general and site-specific aspects of the copper smelting industry were prepared, including the production and emission histories and characteristics. The key emissions were determined to be sulfur dioxide and particulates containing numerous trace metals including lead, arsenic, cadmium, and copper. The industry generally recycles its water so that there are no obvious effluent problems, although indirect contamination of water supplies may be possible from tailings ponds or surface leachates which contain industry-related pollutants.

d. *Etiological Basis*

Research into the etiology of diseases likely to be associated with copper smelting was conducted through literature searches and consultations with medical experts (see Appendix C). Toxic agents produced by the copper industry which have been linked to diseases are summarized below:

- *Sulfur Oxides*—Aggravation of respiratory diseases including asthma, chronic bronchitis, emphysema; eye irritation; suspected in respiratory and gastrointestinal cancers; dystrophic liver functions suspected.
- *Arsenic*—Poorly differentiated epidermoid bronchogenic carcinoma; skin cancer; possibly colon cancer and/or neurological disorders.
- *Lead*—Lead colic; anemia; psychotic states; peripheral neuropathy; may be nephrotoxic.
- *Cadmium*—Correlated with hypertension, liver disease, bone disease, kidney impairment, spleen and thyroid dysfunction.
- *Manganese, Zinc*—Congenital abnormalities.
- *Nickel*—Dermatological irritation; cancer of the sinuses; hemophagic pneumonia (nickel carbonyl).
- *Copper*—Orally causes nausea, vomiting and diarrhea; inhaled causes copper fume fever.

In general, there does not appear to be a strong linkage between the emitted toxic agents and digestive diseases, although both sulfur dioxide and cadmium have been implicated to some extent in liver disease.

e. Collection of Data

Industry Data—Age, production, and emission characteristics were collected for all smelters. Emission data included stack height and control history and the amount of sulfur dioxide (SO_2) and particulate matter discharged. Most plants installed acid plants for the control and recovery of SO_2 emissions in the early 1970's. However, one plant (ASARCO) does not have any controls and uses the technique of curtailment and an extremely tall stack (1,000 feet) to satisfy state pollution regulations.

Air and Water Quality Data—Air quality data were obtained from the State Air Quality Reports for all smelter towns back to 1969 or 1968. These data consisted of SO_2 and particulate matter concentrations. The measured constituents of the particulate matter were benzene solubles, nitrates, sulfates, and most of the trace metals, of which arsenic, cadmium, copper, and lead were the most significant. The key problem with the air quality data was the lack of data for most of the nonsmelter towns of interest, i.e., the control towns. Water quality data were collected for the Gila and Pinal County sites in Arizona from the State Water Control Bureau. Water chemistries were obtained for the public wells for 20 to 30 years at 3-year intervals. The data included the concentrations of minerals and trace metals, but in general it was spotty. No water quality data was obtained from smelter sites other than those in Gila and Pinal Counties in Arizona.

Meteorology/Hydrology Data—Data on wind patterns and inversion characteristics were obtained for the several sites from state and local officials. Qualitative hydrologic characteristics were obtained for each area, including information on water sources for the potable water systems.

Health Data—Mortality data for all diseases were collected for all eight sites investigated. The usual form of data was magnetic tape; however, for the hypothesis development sites in Ginal and Pinal Counties of Arizona, hard copies of the death certificates were also obtained and coded. The hard copy was needed to acquire occupational information and to obtain data prior to 1968. The years of mortality data obtained were as follows:

1957-1975: Miami, Hayden, San Manuel (Arizona)
1968-1975: Ajo, Morenci, Douglas (Arizona)
1968-1975: Anaconda (Montana)
1966-1975: Magna (Utah)

No quantitative morbidity data were acquired. In Gila and Pinal Counties, the hospitals were all owned or influenced by the copper companies and would not release the health information until stronger evidence could be provided that the smelters were the source of community disease. No attempt was made to acquire morbidity data in the other sites investigated, due to a lack of sufficient time to process these data.

Qualitative health information was provided by hospital and state health officials. For example, it was observed by at least two hospital officials that alcohol abuse was a problem in the smelter towns in Gila and Pinal Counties. This was attributed to the ready availability of money supplied by steady copper smelting employment and the lack of cultural activities or other means of entertainment. No health officials had observed any unusual incidence of digestive diseases other than alcoholic cirrhosis of the liver.

Body burden data on lead, arsenic, and cadmium in Arizona were available from recent research performed by the Center for Disease Control. These data were used in developing and testing the causal hypotheses in Arizona.

Demographic Data—Data on population and socioeconomic characteristics were obtained for Arizona, Montana, and Utah from the Census Bureau. Fifth count, file C, tapes from the 1970 Census, which provided the data at the level of enumeration district, were used. Enumeration district maps were acquired to enable the correlation of the street address and town of residence to enumeration district.

f. Analysis of Data

The basic method of analysis consisted of dividing the mortalities into 34 disease categories, and then comparing the age-adjusted mortality rates for the towns hypothesized as being exposed to the rates for the towns assumed to be unexposed, i.e., the controls. Since most towns are a considerable distance apart and, in general, there is no intervening population to speak of, it was a relatively simple matter to separate the population into potentially exposed and unexposed categories. Because the exposed populations for each site were relatively small, various aggregations of towns were examined to facilitate statistical significance testing. The Mantel-Haenszel chi-square test was used to test the statistical significance of the relative risk values for the exposed population.

The mortality data were examined in the light of air and water quality data or body burden data to associate excess mortalities with particular chemicals found in the air, water, or body. In this way, hypotheses were developed at particular sites and tested at others. Attempts were made to examine the effect of duration of residence; however, there were insufficient mortality data to permit any significant findings. To avoid possible confounding by occupational exposure, the principal findings were based on mortalities of females; the copper smelting industry has not employed women in the smelters until the past two or three years.

In Arizona, it was difficult to find suitable control populations whose socioeconomic characteristics matched those of the population at risk. The smelter towns had a different socioeconomic structure than the nonsmelter towns. For this reason, the population of the entire state was used as the control.

Due to the lack of suitable control areas, for which air and water quality or body burden data were available, a dose-response type of analysis was performed with the Arizona data. The approach here was to correlate the concentration of a particular pollutant with the mortality rates for the smelter towns and for the towns near smelters for which data were available. To assist in this correlation and to provide assurance that the resulting correlations were not due to socioeconomic confounding, a factor analysis was performed with all the data for the towns consisting of mortality rates, socioeconomic characteristics, air quality data, and industry characteristics such as age, production, and emission characteristics.

g. Summary of Findings for Copper Smelting Case Study

(1) Causal Hypothesis Development

From the study of the eight copper smelter sites, four causal hypotheses were developed between the industry and four separate diseases, namely, respiratory disease, digestive disease, cerebrovascular disease, and cancer of the colon and pancreas. Of these four diseases, the first two had been correlated with the copper industry by means of the multiple regression analysis; the last two had not.

(2) Respiratory Disease Findings

A strong correlation was found between the ambient levels of SO_2 and particulates and acute respiratory conditions such as pneumonia, influenza, pleurisy, pulmonary congestion, and lung edema. This correlation was demonstrated in two different ways. First, in the matched-control analysis for all smelter sites in Arizona, Montana, and Utah, excessive age-adjusted mortality rates in females were associated with excessive levels of SO_2 and particulates, and lower mortality rates were associated with lower levels of SO_2 and particulates. Second, for 11 towns in Arizona (including some nonsmelter towns) for which air quality and mortality data were available, a factor analysis of the data combined with a multiple regression analysis showed that the mortality rate for acute respiratory diseases in females was strongly correlated with the level of SO_2 and the age of the smelter. The same analysis showed that the disease was not correlated with socioeconomic factors and that no other disease was correlated with SO_2.

These findings, coupled with the well-established relationship between SO_2 and respiratory disease, suggest that SO_2 emissions from the copper smelters cause excessive mortalities from acute respiratory diseases.* It appears that particulate emissions could also be a key factor; however, there is no way to determine at this time the relative importance of SO_2 and particulates in contributing to the deaths, since the two occur together for the sites examined (this is a feature of several SO_2 air pollution epidemiological studies).

The chronic respiratory diseases of bronchitis, emphysema, and asthma were not found to be associated with SO_2 and particulates. This is an interesting finding that deserves some explanation, since the etiology of respiratory diseases has demonstrated that SO_2 is an etiologic agent for these chronic respiratory diseases. A possible explanation is that individuals afflicted with chronic bronchitis, emphysema, or asthma leave the copper smelting areas when the disease develops to a serious level.

(3) Digestive Disease Findings

Digestive and respiratory diseases were the two strongest correlations with the copper industry resulting from the original regression analysis. However, the digestive disease correlation is of special interest because it survived the top 10 county matched-control test and the respiratory disease correlation did not.

Mortality rates in females for diseases of the liver, pancreas, and gallbladder were found to be significantly elevated in the Arizona and Montana smelter towns but not in Utah. No conclusive evidence could be developed that linked the elevated mortalities to a particular pollutant from the copper smelting industry. The most reasonable explanation is that the most prevalent digestive disease (liver cirrhosis) is a result of excessive consumption of alcohol in the copper smelting towns. The excessive use of alcohol is a problem recognized by health officials in Arizona. It is a common malady in rural communities whose residents have relatively high and steady incomes and limited means of entertainment. This relationship is an indirect socioeconomic relationship with the industry that doesn't involve an industrial pollutant. This hypothesis is supported by the following:

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*There are no other large sources of SO_2 in the region.

(a) The health officials in Arizona smelter areas have observed that there is excessive consumption of alcohol by smelter workers and their families.

(b) The smelter counties in Arizona and Montana consume a higher than average quantity of alcoholic beverages.

(c) In Utah, where digestive diseases were not significantly elevated, the majority of residents are nondrinking Mormons.

Lead, cadmium, and arsenic are known to accumulate in and cause damage to the liver. The levels of these heavy metals are elevated in some smelter areas; however, the levels cannot be correlated with digestive disease prevalence to any significant degree. Although a direct effect or a synergistic effect with alcohol cannot be ruled out, it appears that copper smelter pollutants are not a major factor in the elevated digestive diseases found in many copper smelter communities.

(4) Findings for Other Diseases

Mortality rates in females for cerebrovascular disease and cancer of the colon and pancreas were significantly elevated in Anaconda in Montana but not in Arizona or Utah. Similarly, levels of cadmium and arsenic were very high in Anaconda but not in the average Arizona smelter town (data for Utah was not available). Considering the etiology of the diseases, these findings could be explained by the following hypotheses:

(a) The high levels of cadmium and arsenic (a suspect human carcinogen) emitted from the smelter in Anaconda are a possible cause of excess mortalities from cancer of the colon and pancreas.

(b) The high levels of cadmium are a possible cause of the excess mortalities from cerebrovascular disease.

It is noted that both the disease etiologies and the supporting data are not sufficiently strong to provide convincing evidence of a possible relationship between the diseases and the industry. These relationships are highly speculative at this time, and considerable additional evidence is needed to provide a credible causal hypothesis.

3. *Steel Manufacturing Case Study*

a. *Basis for Correlation*

The multiple regression analysis found a fairly strong correlation between an industrial grouping that contained steel manufacturing, and cardiovascular diseases and digestive neoplasms in females. Close inspection of the data disclosed that steel manufacturing was the main component of the industrial group. Upon screening the correlation using a matched-control test for the top eight steel manufacturing counties, the cardiovascular correlation disappeared in females. However, the correlation with digestive neoplasms in females remained strong. Since steel plants were not expected to employ females in hazardous areas, it was assumed that the validated correlation between steel manufacturing activity and digestive neoplasms in females was an indication of a community health problem and, consequently, this correlation was selected for investigation.

b. *Study Site Selection*

Johnstown, Pennsylvania, was selected as the site of the initial field investigation for the purpose of developing a causal hypothesis. There were several reasons for selecting Johnstown:

- high industrial index for steel manufacturing;
- high county mortality rate for digestive neoplasms in females;
- relatively large population at risk;
- excellent data available, including mortality data on tape, morbidity data from a county registry, and fairly complete air quality data;
- no other large industries that might confound the findings.

Provo, Utah, was selected as the hypothesis test site on the basis of high industrial index, little industrial clutter, large population at risk, and good availability of data including morbidity data on tape from the cancer registry.

It is emphasized here that, in selecting sites for the steel investigation and, indeed, in selecting the sites for all the investigations, a prime consideration was the avoidance of the potentially confounding factors associated with large cities such as multiple sources of pollutants and extensive urbanization. Hence, large metropolitan areas were not considered in the search for study sites. In the case of steel, this meant that the large steel cities of Pittsburgh, Buffalo, and Birmingham were not considered suitable sites for the present investigation.

c. *Industry Characterization*

A full description of the general and site-specific characteristics of the steel industry pertaining to possible community health problem was prepared. In general, a steel plant is comprised of four major processing units: coke ovens which produce coke for firing the furnaces, sintering plants which process fugitive metal fines into reusable form, blast furnaces which produce iron from ore, and steel-making furnaces (open hearth, electric arc, or basic oxygen) which produce steel from pig iron and scrap. Coke ovens emit particulates and organics including benzo[a]pyrene (BaP). Sintering plants emit hydrocarbons and particulates. Blast furnaces and steel furnaces emit large quantities of particulates and oxides.

d. *Etiological Basis*

The organics, hydrocarbons, and particulates produced by the various steel processes have a recognized potential for causing disease. Laboratory and occupational health research has associated a number of diseases with the emissions from steel plants. Summarized below are the findings related to etiological agents and associated diseases:

- *Organics (includes BaP)*—malignant neoplasms (including digestive neoplasms); respiratory disease; nephritis; gastroenteritis; liver disease.
- *Sulfur Dioxide*—respiratory disease; digestive and respiratory neoplasms; liver disease.
- *Particulates*—digestive and respiratory neoplasms; respiratory disease.

- *Lead*—gastrointestinal and respiratory diseases; central nervous system diseases.
- *Cadmium*—hypertension and other cardiovascular disease; liver disease; nephritis.

As is evident from this listing, there is a substantial etiological basis for a cause-and-effect relationship between the steel industry and digestive neoplasms. Organics, sulfur dioxide, and particulates produced by steel processes have been related to neoplasms of the digestive organs.

e. *Collection of Data*

Industry Data—Data on the age, production history, and total emissions of criteria pollutants were assembled for the steel plants under study. An interesting method of obtaining and verifying information on the growth of the steel industry in Johnstown was utilized. By analyzing several sets of aerial photographs taken over the past 40 years, the EPA Environmental Photographic Interpretation Complex (EPIC) in Warrenton, Virginia, was able to determine when new plants were built or operations were expanded. Also, EPIC was able to verify that there were no large point sources of pollution in the Johnstown Air Basin other than the steel industry.

Air and Water Quality Data—Excellent air quality data for Johnstown were obtained from the Pennsylvania Department of Environmental Resources. Several years of data for criteria pollutants, trace metals, BaP, and benzene-soluble organics (BSO) were available from monitoring stations in the Johnstown Air Basin. The air quality data for Provo, Utah, and vicinity were not as complete as we had originally been led to believe. The principal data obtained was for total suspended particulates (TSP). For both Utah and Johnstown, some data were obtained from an EPA study which provided BaP and TSP measurements. It was determined that communities in the vicinity of both Johnstown and Provo obtained their potable water from sources too remote to be affected by effluents or emissions from the steel plants.

Meteorology Data—Air basin characteristics for Johnstown in Pennsylvania were obtained from Pennsylvania State University studies of the area.

Health Data—For both the Johnstown and Provo sites, mortality data for all diseases were obtained on magnetic tapes for 11 years, from 1965 through 1975. The number of mortality records obtained is given in Table II-3. These data did not contain duration of residence or occupational information. Morbidity and mortality data from 1966 to 1975 were also obtained for the Provo site from the Utah Cancer Registry. A problem with these data was that they provided incidence and prevalence by county and not by town or census unit. We had planned to obtain and analyze cancer registry information for Johnstown also, but the flood of July 1977 prevented this. An interesting characteristic of the Provo site is that the majority of the population is Mormon; thus, health problems related to alcohol or smoking are less prevalent.

Table II-3. MORTALITY RECORDS ACQUIRED FOR STEEL INVESTIGATION

	Number of Deaths from All Diseases for All Ages			
	Exposed Areas		Control Areas	
	WM	WF	WM	WF
Johnstown Site	4,000	3,300	5,300	3,700
Provo Site	4,000	3,300	4,400	3,300

WM = white males; WF = white females

Demographic Data—The fifth count, file C, tapes from the 1970 Census were used for population and socioeconomic data. This file provides data at the enumeration district level or the census tract level.

From the analysis of aerial photographs, EPIC determined key population shifts over a 30-year period for the Johnstown area. Using meteorological and topographic data, EPIC developed estimates of the population at risk as a function of time for the Johnstown Air Basin.

f. Analysis of Data

To develop a causal hypothesis at the Johnstown site, exposed and control populations were selected on the basis of air transport and socioeconomic considerations. The work of EPIC was used to assist in the definition of the exposed population. A control population that matched selected socioeconomic variables to within 10% was selected.

As in the case of the copper smelter case study, mortalities were divided into 34 disease categories, and the age-adjusted mortality rates of the exposed population were compared to those of the control population. The significance of differences in mortality rates was computed using the Mantel-Haenszel χ^2 test. Causal hypotheses were developed by correlating differences in mortality rates between exposed and control populations with differences in air quality measurements, and identifying those correlations having sound etiological basis.

Although both male and female mortality rates were computed, to preclude bias due to occupational exposure, the hypotheses developed were based only on the data for females. Male mortality rates were examined to provide verification where possible, and to see if unusual health problems of possible occupational origin were indicated by the data.

At Provo, Utah, this process was repeated with special attention given to the diseases and specific emissions identified from the Johnstown analysis. The exposed population consisted of industrial towns or municipalities, which were examined separately rather than by aggregation. Control populations were found that matched the selected socioeconomic variables to within 15% or less.

g. Summary of Findings for Steel Manufacturing Case Study

(1) At the Johnstown site, there are a number of diseases with significantly elevated age-adjusted mortality rates in females. These are:

- Malignant neoplasms
    - —oral cancers (males & females)
    - —respiratory cancers (males & females)
    - —breast, urinary, genital cancers (males & females)
    - —central nervous system cancers (males & females)
    - —peritoneal and other digestive cancers (females only)

- Cardiovascular disease (males & females)
- Respiratory disease (females only)
- Urinary disease (females only)

For all other diseases, there were no significant differences between the exposed and control death rates in females.

(2) No conclusive evidence was obtained which linked a specific pollutant to any of the elevated diseases. However, on the basis of etiological considerations and of the measured high levels of pollutants in the exposed areas compared to national standards, the following hypotheses were indicated:

Disease	*Etiologic Agent*
Malignant neoplasms (oral, respiratory, breast, CNS, peritoneal)	Organics, particulates
Cardiovascular disease	Cadmium
Respiratory disease	Organics, particulates, SO_2, lead
Urinary disease	Organics, cadmium

The organics emissions (e.g., BaP) are the predominant etiologic agent in these hypotheses taken as a whole. Hence, the primary causal hypothesis developed is the relationship between organic emissions from the coking processes and a variety of diseases in the community, principally cancers. This is in agreement with the original correlation from the multiple regression analysis which correlated digestive cancers with the steel industry.

(3) At Johnstown, a number of additional diseases were found to be significantly elevated in males only, indicating that they possibly were of occupational origin. These diseases are: stomach cancers; leukemia; blood diseases; hypertension; cerebrovascular diseases; bronchitis, emphysema, asthma; liver diseases. No checks could be made to verify that these diseases were occupationally derived; data and time did not permit this.

(4) Only one of the causal hypotheses developed at Johnstown was supported by the Provo data. Mortality rates for malignant neoplasms of the breast, urinary, and genital organs and of the central nervous

system were found to be significantly elevated at Provo as well as at Johnstown. Other neoplasms and cardiovascular, respiratory, and urinary diseases were not significantly elevated in Provo.

(5) It was determined that the pollutant concentrations of organics and particulates at the Utah site are at least an order of magnitude less than the concentrations at Johnstown. This is due to differences in plant emission concentrations, proximity of the exposed population to the pollutant sources, and meteorological and topographic characteristics. The latter is a particularly strong reason for the difference: the Johnstown site is in a narrow and well-defined air basin, which traps the emissions. At Provo, the population is located in a very wide valley where emissions are not confined. Another contributing factor to the difference is the fact that the Johnstown Steel Works are at least 50 years older than the Geneva Works; hence, the Johnstown population has had a longer exposure to pollutants than has the Provo population. These factors suggest that some of the causal hypotheses identified in Johnstown may be valid, even though the death rates for the diseases are not elevated in Provo. For example, respiratory cancers at Provo were found to be elevated in males but not in females. The high rates in males could be due to occupational exposure, and the normal rates in females could indicate that the community exposure is too low to induce a measureable effect.

(6) In both sites, the death rates from endocrine, metabolic, and blood diseases were found to be elevated in males and not in females. This may indicate an occupational problem possibly caused by exposure to trace metals within the plant.

4. Bituminous Coal Mining Case Study

a. Basis for Correlation

The strongest correlation produced by the multiple regression analysis was between bituminous coal mining and cardiovascular and respiratory diseases. The correlation was strong in both males and females, indicating a potential community health problem. The correlation passed all of the screening tests, including the matched-control test for the top 10 coal mining counties.

b. Study Site Selection

Wyoming County, West Virginia, was selected as the causal hypothesis development site because of high industrial activity, lack of confounding industry, high mortality rates for the diseases of interest, and availability of morbidity data.

For the hypothesis test site, the coal mining area of southern Illinois (counties of Saline, Williamson, Franklin, Randolph, and Perry) was selected. This area was chosen principally because of high industrial activity and the potential of relatively large exposed and control populations. Also, there was no appreciable industrial clutter and mortality data were readily available.

c. Industry Characterization

The three principal elements of coal mining operations are the mine, the coal preparation plant (tipple), and the coal transportation system (i.e., track conveyors, trucks, and railroad cars). In Wyoming County, underground mining is predominant; in southern Illinois, both surface and underground mining are prevalent. Coal preparation plants clean and size the coal. Usually water is used in the cleaning process, and the coal is dried using shakers, centrifuges, or thermal dryers. Thermal drying, accomplished by heated air, is the predominant drying process in Wyoming County and southern Illinois.

All coal operations produce pollutants to some degree. The three principal pollutants are acid mine drainage, coal dust emissions, and coal combustion products. Mines and coal preparation plants discharge water that is often highly acidic (acid mine drainage) and capable of contaminating community water supplies with solubilized toxic minerals. Coal dust is discharged by mines and coal preparation plants, principally thermal dryers. Also, coal handling, storage, and transportation in and around coal preparation plants produce significant quantities of fugitive coal dust emissions. Coal combustion products are produced principally by thermal dryers which burn coal to generate the hot flue gases for drying. Also, coal combustion gases are emitted from burning piles of waste (gob piles) discharged by the coal preparation plants.

In recent years (since the late 1960's), the thermal dryers have incorporated highly efficient control devices to reduce the particulate emissions. However, combustion gases are not controlled and EPA believes that the amount of NO_x and SO_x emitted is too small to require control. Although coal dust emissions from coal handling, storage, and transportation could largely be controlled by enclosing the operations or covering the coal with tarpaulins, there does not appear to be much of this in practice.

d. Etiological Basis

The health hazards associated with coal mining occupations are well recognized and documented. There are four major occupational respiratory diseases of coal miners that have been linked with coal dust and related emissions:

- *Coal Workers' Pneumoconiosis* (simple CWP)—this results from the accumulation of inhaled coal dust in discrete macules in the lung and can be associated with some changes in respiratory function; rarely if ever disabling.
- *Progressive Massive Fibrosis* (PMF, or "complicated" CWP)—this is an advanced, uncommon form of CWP which results in obstructive (emphysematous) and restrictive pulmonary disease and may be life threatening.
- *Chronic Bronchitis*—this probably represents chronic bronchial irritation from dust, but it has not been extensively studied.
- *Silicosis*—this occurs in workers who drill rock and sink shafts through free silica (SiO_2) deposits or who drive the transport cars and can be life threatening in severity.

With regard to cardiovascular diseases associated with coal mining, an excess mortality from heart disease is a predictable result of a high prevalence of lung disease. The pulmonary and cardiovascular systems are intimately related, and there exist several mechanisms by which lung dysfunction can result in heart failure. Other environmental factors such as low water hardness have been associated with cardiovascular disease; however, mine drainage would tend to increase rather than decrease water hardness.

e. *Collection of Data*

Industry Data—Data on location, age, production, and other characteristics of the special mining operations at the sites were obtained from Keystone Coal Buyers Manuals. Source emission data were obtained for the counties from the National Emissions Data System (NEDS).

In Wyoming County, West Virginia, there are many underground mines and several preparation plants, five of which have thermal dryers. In southern Illinois, there are both surface and underground mines and ten preparation plants, five of which have thermal dryers. Actual numbers of mines and preparation plants vary considerably over time. Because of the relatively small population that may be exposed to an individual mine or preparation plant, it was necessary to examine a number of mines or plants at each site in order to provide large enough exposed populations. In effect, this treated the industry as an area rather than as a point source.

Air and Water Quality Data—No air quality data were available for either site; air quality had not been monitored in any of the counties by the states or counties. In the absence of air quality measurements, dispersion models were used to estimate concentrations of pollutants in communities. Water chemistry data for Wyoming County potable water supplies were obtained from the County Health Department.

Meteorology/Hydrology Data—The two sites investigated are very different in topography and weather. The Wyoming County site consists of narrow valleys which confine the emissions and effluents to relatively small regions. The southern Illinois area is flat in comparison (rolling hills), enabling wide dispersal of the emissions and effluents.

The needed meteorological data for analysis were obtained from the closest weather stations in both areas. General hydrologic information was obtained from state officials; site-specific hydrologic information was obtained by on-site observations.

Health Data—Death certificates for respiratory and cardiovascular diseases for Wyoming County in West Virginia for the years 1968-1972 were supplied by the West Virginia Vital Statistics Office; death certificates for all diseases for the years 1965-1975 were ordered but never received. Mortality data for the Illinois site were obtained for all diseases for the years 1974-1976.

Morbidity records for all illnesses were obtained from Wyoming General Hospital in Wyoming County. These data were abstracted by local medical record abstractors. Diagnostic and demographic data were obtained for all diseases and the cardiovascular and respiratory cases were also staged for severity, using a form designed for this purpose. The plan had been to obtain mortality records from two other hospitals in the area which, together with Wyoming General, would have provided an estimated 75% of the admissions from Wyoming County; however, approvals could not be obtained in time to acquire these data. Consequently, the morbidity records that were collected from Wyoming General are of limited value due to admission selectivity.

Some qualitative observations related to health problems in Wyoming County were offered by hospital staff members. They observed that coal miners are heavy tobacco users, smoking cigarettes when they are out of the mines and chewing tobacco in the mines where smoking is not allowed. It was estimated that better than 90% of the miners use tobacco extensively, and this presently appears to be extended to their wives. This indicates that tobacco use is a factor to be considered in any findings.

Demographic Data—Population and socioeconomic characteristics were obtained from the fifth count, file C, 1970 Census tapes, which provide data at the enumeration district level. Enumeration district maps were acquired to enable the correlation of the street address and town of residence to enumeration district.

f. Analysis of Data

Correlating etiological findings with the mining-related emission and effluent characteristics, several conceptual causal hypotheses were derived for Wyoming County. These include:

- Respiratory/cardiovascular diseases caused by coal dust emissions from mining operations, principally the operations associated with coal preparation plants utilizing the thermal drying process.
- Respiratory/cardiovascular diseases caused by coal combustion products from thermal dryers and burning waste piles.
- Cardiovascular diseases caused by the toxic effects of acid mine drainage (e.g., solubilized cadmium).

By analyzing the water quality and the transport of emissions, assessments were made of the plausibility that Wyoming County communities would be exposed to hazardous levels of hypothesized etiologic agents. Age-adjusted mortality rates for cardiovascular and respiratory diseases were then computed for potential populations at risk and compared to control populations. To assist in determining the part that terrain and socioeconomics play in the relationship between industry and disease, a regional analysis was made of the county-by-county mortality and industry data that had been used as the data base for the initial regression analysis.

The hypothesis developed by integrating the results of the emission transport, water quality, and mortality analyses was then tested by two separate investigations: first, by the analysis of morbidity data (hospital patient records) in Wyoming County; and second, by a mortality analysis at the southern Illinois site. In both cases, disease patterns were examined to determine if they supported the hypothesis. At the Illinois site, an emission transport analysis was performed to estimate the level of exposure to the population at risk.

g. *Summary of Findings for Bituminous Coal Mining Case Study*

(1) In Wyoming County and southern Illinois, the respiratory and cardiovascular mortality rates for communities in the proximity of thermal dryers exceeded the rates for distant populations. The amount of excess was statistically significant for all cases except for respiratory diseases in Illinois, where the rate ratio of 1.5 was not statistically significant.

(2) Based on the water quality analysis, hazardous levels of toxic minerals were not found in Wyoming County water systems. The evidence indicates that acid mine drainage is not a health problem in Wyoming County.

(3) The analysis of Wyoming General Hospital records in Wyoming County disclosed no unusual morbidity patterns. This result is not particularly significant, since only approximately one-fourth of the morbidities in Wyoming County were analyzed and the hospital selectivity bias for those examined is unknown.

(4) The results of the regional mortality analysis show that, when the mortality rates for respiratory and cardiovascular diseases are compared for coal-mining and non-coal-mining counties in similar terrain, the rate ratios are higher in mountainous terrain than in flat terrain, indicating that terrain is a factor in the relationship between coal mining and respiratory/cardiovascular diseases.

(5) Although there is no conclusive evidence of the existence of hazardous levels of coal dust in the coal mining communities, the results of the investigation point towards the hypothesis that coal dust from operations in and around the thermal dryer coal preparation plant is the cause of the elevated respiratory and cardiovascular disease. The etiology is sound and the results of the site-specific mortality analyses show a correlation between the proximity to thermal dryer coal preparation plants and mortality rates. The results of the regional mortality study can be explained by the fact that the narrow valleys in mountainous terrain tend to confine the coal dust to the

populated areas. The results of the morbidity study do not support the coal dust hypothesis; however, as previously mentioned, these results are discounted due to the unknown hospital selectivity bias.

(6) Some consideration was given to the socioeconomic hypothesis that heavy smoking by coal miners and their families and the use of coal for home heating are the causes of excess respiratory and cardiovascular diseases. Although these hypotheses are not supported by the evidence produced, the contrary evidence is not sufficiently strong to rule them out.

5. *Viscose Rayon Case Study*

a. *Basis for Correlation*

The basis for the correlation between the viscose rayon industry and community disease, principally coronary heart disease, differs from the other case studies. The viscose rayon correlation is not a result of the multiple regression analysis, as were the others; rather, it is a result of community complaints regarding an odor problem associated with a particular plant. Upon investigation, it was ascertained that potential toxicants (hydrogen sulfide and carbon disulfide) are emitted in substantial amounts from the industry and that there is documented evidence that these toxicants may be harmful to viscose rayon workers. A comparison of the mortality rates between counties with viscose rayon plants and control counties showed that coronary heart disease was significantly elevated in some of the exposed counties.

b. *Study Site Selection*

Two sites were selected for field investigation on the basis of the history and characteristics of the viscose rayon plant, the size and proximity of the population at risk, the absence of other industry and pollution sources, the availability of emission and air quality data for the pollutants of interest, and the availability of health data. The two sites were Elizabethton, Tennessee, and Lewistown, Pennsylvania.

c. *Etiological Basis*

A search of relevant medical and occupational health literature disclosed that there is considerable evidence that elevated coronary heart disease and death due to cardiovascular disease in viscose rayon workers are associated with exposure to carbon disulfide (CS_2) and hydrogen sulfide (H_2S) in workplace air. A study of rayon manufacturing processes and plants disclosed that relatively large quantities of CS_2 and H_2S are emitted to the atmosphere during the manufacturing process. Thus, the potential exists for chronic exposure of nearby populations to these etiologic agents.

d. Collection of Data

Various industry, environmental, and health data were collected for the two sites. These data consisted of:

- Plant process characteristics and production history
- Emission estimates and point source data
- Meteorological data
- Air and water quality data
- Demographic data for exposed and control populations
  - fifth count, file C, 1970 Census
- Mortality data for all diseases
  - Elizabethton, Tennessee; 1960-1975 (total deaths, 2,190; deaths 35-74, 1,203 records)
  - Lewistown, Pennsylvania; 1965-1975 (total deaths, 2,992; deaths 35-74, 1,546 records)

e. Analysis of Data

A transport analysis was performed to estimate populations exposed to various levels of CS_2 and H_2S. Two types of controls were used to test the hypothesis at each site. One set of controls consisted of the county residual which consisted of the population of the county living outside the city that contained the rayon plant and considered not to be at potential risk. The other set of controls consisted of an aggregate of towns which were demographically and socioeconomically similar to the study site. Age-adjusted mortality rates for 34 disease categories were computed for males and females and compared for exposed and control populations. Differences in mortality rates were tested for significance by means of the Mantel-Haenszel χ^2 test.

f. Summary of Findings for Viscose Rayon Case Study

(1) No death rates (for any of the 34 disease categories) were found to be significantly elevated in the exposed populations at either Elizabethton or Lewistown. That is, a significant community health effect associated with viscose rayon plants could not be found from the analysis of the mortality data.

(2) Ambient CS_2 concentrations for the exposed populations were estimated to be in the range of 25 to 100 ppb. The plants in both sites have been operating since before 1930. These findings indicate that chronic exposure of populations to these ambient concentrations of CS_2 will not induce premature death.

B. DISCUSSION OF RESULTS

1. Validity of Causal Hypotheses

In three of the four industry-disease investigations, plausible causal hypotheses were derived from the site-specific data and analysis. The principal relationships are summarized in Table II-4.

This section discusses the validity of these hypotheses in terms of five specific measures of merit:

- coherency of explanation
- strength of association
- specificity of association
- time sequence of variables
- consistency of association

These five measures of merit are summarized for each of the relationships in Table II-5 and are discussed in the paragraphs below.

a. Respiratory Diseases Associated with Copper Smelting

This is the most well-founded relationship developed during the study. It scores satisfactory marks on all five measures of merit. Particular features are the strength of the relationship borne out by both a matched control and a dose-response type test, the consistency of the association in several study sites, the temporal relationship with the age of the smelter, and the lack of a correlation of SO_2 with other diseases and socioeconomic factors. The key weaknesses are the lack of verification of the duration of exposure for the decedents and the deficiency in strength due to the small exposed populations and the small number of observations. Additional work that is needed would consist of the following:

- Verification of the exposure of decedents by establishing the duration of residence from state and local records.

- Collection and analysis of hospital records to further strengthen the association and investigate the hypothesis that the incidence of obstructive pulmonary diseases is also elevated but does not result in mortalities because those with the disease move away from the smelters and

Table II-4. *PRINCIPAL CAUSAL HYPOTHESES*

INDUSTRY	DISEASE	ETIOLOGIC AGENT	REMARKS
Copper Smelting	Acute Respiratory Diseases (pneumonia, influenza, pleurisy, etc.) 1	SO_2 emitted from smelters Particulates may be contributing agent	Chronic inhalation of SO_2 and particulates damages pulmonary system which decreases resistance to acute respiratory diseases
	Digestive Diseases (primarily cirrhosis of the liver) 2	Alcohol principally Cadmium emitted from smelters a possible contributing agent	Excessive consumption of alcohol in copper smelting community causes high prevalence of liver cirrhosis mortalities
Steel Manufacturing	Malignant Neoplasms 3	Organic emissions (e.g., BaP) from coking and smelting plants Particulates from various processes also a contributing agent	Inhalation and ingestion of organics and particulates induce malignant neoplasms in various organs
Bituminous Coal Mining	Respiratory/Cardiovascular Diseases 4	Coal dust emitted from coal mining operations, principally those in and around thermal dryer coal preparation plants	Inhalation of coal dust leads to pulmonary dysfunction which leads to cardiac involvement

Table II-5. *SUMMARY EVALUATION OF PRINCIPAL DERIVED CAUSAL HYPOTHESES*

	ASSOCIATION	COHERENCY OF EXPLANATION	STRENGTH OF ASSOCIATION	SPECIFICITY OF ASSOCIATION	TIME SEQUENCE OF VARIABLES	CONSISTENCY OF ASSOCIATION
1	Acute diseases of respiratory system associated with copper smelting through SO_2 & particulate emissions.	Well-established etiology between SO_2 & respiratory diseases. Reasonable explanation can be given for why acute diseases were found to be associated & chronic respiratory diseases were not.	• Matched control test showed statistically strong correlation between disease & industry. • Dose-response test showed strong correlation between SO_2 concentrations, age of industry & disease.	• Specific disease & specific etiologic agent have been identified. • Other diseases not correlated with agent. • Elevated mortality rates found in nonoccupational portion of population (females) indicating community disease. • Socioeconomic factors not correlated with mortality rates.	• Estimated period of exposure consistent with mortality data. • Emission measurement data consistent with exposure period. • Mortality rates shown to be correlated with age of smelters. • Could not determine period of exposure of individuals who died of disease.	• Association validated by both matched-control and dose-response type of test. • Association consistent between copper smelting sites.
2	Digestive diseases, primarily alcoholic cirrhosis of the liver, associated with copper smelting	No coherent explanation why copper smelting emissions or effluents would induce high digestive disease mortality rates, particularly for liver cirrhosis. Evidence that cadmium emitted by smelters could be a contributing cause; however, the most logical explanation, supported by alcohol consumption data & health observations, is the abuse of alcohol in copper smelting areas.	• Strong correlation between disease & industry, based on matched-control test, except in Utah. • Recognized (but not verified) correlation between alcohol consumption & copper industry, except in Utah.	• Specific disease & specific etiology identified (alcoholic cirrhosis of liver). • Disease prevalence occurring in females, i.e., nonoccupational association with copper smelters.	• Disease prevalence data consistent with age of plant. • No specific data or analysis showing temporal trends & time sequences.	• Association consistent between sites.
3	Cancers of oral, respiratory, digestive, breast & genitourinary systems associated with steel manufacturing through organic emissions from coking plants.	Well-established etiology relating malignant neoplasms to organic emissions, which are produced by coking and sintering plants.	• Strong correlation between diseases & industry based on matched-control test in Johnstown. • Measurement data show exposed population in Johnstown exposed to levels of organics much higher than national levels. • Little correlation between diseases & industry at Provo, Utah. • Measurement data show organic concentrations at Provo are much lower than at Johnstown.	• Rather broad set of diseases associated with a broad category of emissions. • Disease prevalence occurring in females, i.e., nonoccupational association with steel industry. • Diseases other than associated diseases are closely matched between exposed & control areas.	• Estimated period of exposure consistent with disease prevalence data. • Emission measurement data consistent with exposure period. • No specific data or analysis showing temporal trends & time sequences.	• Association not strongly verified at second site; although consistency has not been demonstrated, the lack of association at second site is not necessarily inconsistent & can be explained by the low concentrations observed.
4	Cardiovascular & respiratory diseases associated with bituminous coal mining, through coal dust emissions from thermal dryer coal preparation plants.	Good coherent explanation based on well-established etiology connecting coal dust to pulmonary diseases.	• Correlation between disease prevalence & industry relatively weak because of small exposed population. • No verification that the populations are exposed to hazardous levels of coal dust.	• Specific disease & specific etiologic agent identified. • Specificity is weak because other diseases were not examined.	• Estimated period of exposure consistent with mortality data. • No specific data or analysis showing temporal trends & time sequences.	• Association consistent for two sites but relatively weak in both cases.

die elsewhere. In addition, the health records would provide data on smoking history, which could be used to check the effect that smoking has on the causal relationship.

- Investigation of additional copper smelting sites to further strengthen the relationship. A particularly valuable site would be Tacoma, Washington, where the smelter is located within a city and there are large populations at risk. Analysis of mortality data at the census tract level would permit a dose-response type of analysis as well as a matched-control analysis.

b. Digestive Diseases Associated with Copper Smelters

This is a less well-founded relationship between the disease (liver cirrhosis) and a socioeconomic factor (alcohol consumption) associated with the industry. The association is founded principally on the correlation between the prevalence of liver cirrhosis and the presence of the industry. A specific agent (alcohol) has been identified, and some evidence has been acquired which associates excess alcohol consumption with copper smelter towns. However, further work is needed to provide more convincing evidence, namely:

- Collection and analysis of alcohol consumption data for copper smelting and control towns.
- Analysis of hospital records to provide verification of the incidence of alcoholic cirrhosis.
- Investigation of additional sites to strengthen the association (see previous remarks on Tacoma, Washington).
- Determination of body burden of cadmium in digestive disease morbidities or mortalities if possible.

c. *Malignant Neoplasms Associated with Steel Plants*

This appears to be a fairly sound relationship based on etiological considerations and the strength of association found in Johnstown, where the concentrations of the etiologic agents were very high. However, the relationship needs confirmation in other sites, since the investigation in Provo, Utah, did not support it strongly, owing perhaps to much lower concentrations of toxicants. Thus, additional work is needed to test and validate the relationship:

- Investigation of additional sites, with and without coke plants to verify that they are the primary contributor to neoplasms. The large steel plants in the large cities would be good sites because of high concentrations and good population characteristics.
- Verification of exposure of deceased individuals by establishing duration of residence from state and local records.
- Collection and analysis of hospital and cancer registry records to obtain and analyze smoking histories of patients.

d. *Respiratory and Cardiovascular Diseases Associated with Bituminous Coal Mining*

Although the investigation provided a good coherent explanation of the association, the strength of the association is not satisfactory due to small exposed populations and a lack of measurements to verify the existence of hazardous concentrations of coal dust in the community. Further evidence is needed to validate the relationship, namely:

- Air quality measurements in exposed and control areas to verify differences in coal dust concentrations.
- Collection and analysis of mortality data for all diseases, and for as many years as possible, to investigate the specificity of the association.

- Collection and analysis of hospital records for all hospitals in region.
- Investigation of additional sites.

e. *Summary of Causal Hypothesis Evaluation*

The four case studies have developed and tested a number of hypotheses with the following results:

- One well-founded and verified hypothesis was developed (respiratory diseases-copper smelting).
- One reasonably well-founded hypothesis was developed which needs verification (malignant neoplasms-steel plants [coking process]).
- Two weak hypotheses were developed which need considerably more data and analysis to provide satisfactory strength and confirmation (digestive diseases-copper smelting; respiratory/cardiovascular diseases-bituminous coal mining).
- One negative investigation (cardiovascular disease-viscose rayon plants) which found no site-specific evidence of a relationship between community disease and the industry.

2. *Validity of Regression Analysis*

What can be said about the validity of the multiple regression approach in light of the results of the four case studies? Table II-6 is a comparison of the results of the original multiple regression analysis with those of the case studies. For all the correlations identified by the regression analysis, plausible causal hypotheses were established from the analysis of site-specific data. For the one correlation that was identified from considerations other than the regression analysis, no causal relationship could be developed from the field data.

On the face of it, these results tend to give considerable credence to the multiple regression analysis of county mortality and industry

Table II-6. *COMPARISON OF RESULTS OF MULTIPLE REGRESSION ANALYSIS TO CASE STUDY RESULTS*

	ORIGINAL CORRELATIONS		RESULTS OF CASE STUDIES	
	Industry	Associated Diseases	Was Causal Hypothesis Developed?	Strength of Evidence
Correlations identified by Multiple Regression Analysis	Copper Mining (copper smelting)	Respiratory Diseases*	Yes	Good
		Digestive Diseases	Yes	Fair
	Steel Manufacturing	Digestive Neoplasms	Yes	Fair
		Cardiovascular Diseases*	Yes	Weak
	Bituminous Coal Mining	Respiratory Diseases	Yes	Weak
		Cardiovascular Diseases	Yes	Weak
Correlation identified by other means	Viscose Rayon	Cardiovascular Diseases	No	Good

* These diseases did not appear to be correlated on the basis of top 10 county matched-control test, however they were correlated in the original regression analysis.

data as a means for identifying sources of community disease. However, this conclusion must be tempered with consideration of the limitations of both the case studies and the regression analysis.

In the first place, the results of the case studies are by no means conclusive. Although evidence was found in all multiple regression cases of plausible causal relationships supported by coherent explanations, in general the evidence is quite weak. Further investigation is needed to provide conclusive evidence in all cases.

Secondly, the negative result for the non-regression correlation may not be particularly significant. It shows that our choice of a potential community health problem was not a good one. Further, it shows that our validation methods based on a matched-control test for the top 10 counties are not particularly effective. As far as the multiple regression analysis is concerned, the only significance that can be attached to the negative result for viscose rayon is that the regression analysis appears to be more effective than our method in identifying real industry-disease associations.

We should also examine the results in the light of what we would expect from the multiple regression analysis. The basic concept, upon which the multiple regression analysis of county data is founded, is that, for communities exposed to disease-causing substances from industry, there exists a relationship between the mortality rate in the community and the chronic dose received. Figure II-1a depicts this relationship. If the dose that the population received and the mortality rates could be observed in many communities, then a regression analysis of these data would show a strong correlation between the industry and the disease, because the variance about the regression line (the mortality rate-dose relationship) is much less than the total variance in mortality rates giving no consideration to the dose received.

However, the data on community mortality rates and dosage do not exist; instead, we must use county mortality rates as an estimate of community rates, and industrial index as a measure of dose. To see what this will do to the correlation, let us first examine the effect of county mortality rates.

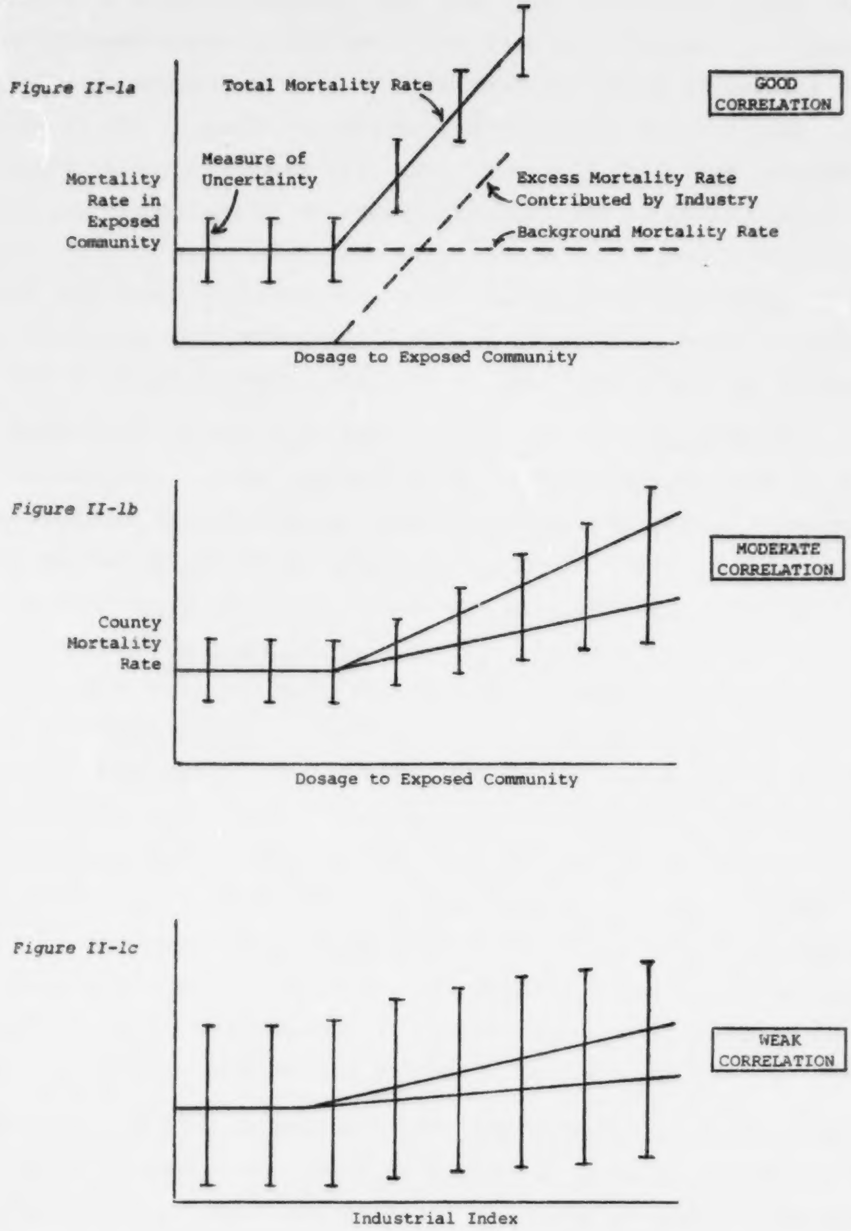

Figure II-1

Correlation Between Mortality Rate, Dosage, and Industrial Index

When county mortality rates are used instead of those of the exposed community, the increases in mortality rate due to the community exposure will be diluted by being included with the entire population of the county. This has the effect of decreasing the slope of the relationships between mortality rate and dose. The amount of the decrease will depend on the ratio of the exposed population to the county population. Since this will vary from county to county, this introduces an uncertainty as shown in Figure II-1b. Here, the variance about the regression line is large compared to the total variance, and therefore the correlation is much weaker than in the ideal case of Figure II-1a.

The surrogate measure for dose is the industrial index.* Dose is a product of time of exposure and concentration, where concentration is a function of source strength, transport phenomena, and distance of exposed community from industry. The only one of these factors that is related to industrial index is the source strength; hence county-to-county variations in time of exposure (age of plant and length of residence), transport phenomena, and distance from the plant will introduce equivalent uncertainties in the relationship between industrial index and dose. Furthermore, the relationship between industrial index and source strength is both weak and uncertain. Variations in type of plant, processes, plant management, and controls introduce uncertainties. The relationship is also diluted in the same way as mortality rates due to the fact that the industrial index is a measure of the industrial activity in a four-digit SIC code, which often is a broad industrial category containing several different types of industries in addition to the type which is producing the harmful substance.

Figure II-1c illustrates how the relationship will be affected by using industrial index as a measure of dose. Here, the variance about the regression line is nearly as large as the total variance; hence, a very weak correlation would result from the regression analysis.

From these considerations, we would expect the correlation coefficients between county mortality rates and industrial indices to be quite small, and indeed they were. However, this alone would not preclude an effective multiple regression analysis, because the analysis of a large number of observations (3,000 counties) could easily pull out the correlation even though there is a great deal of dilution and uncertainty.

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*Industrial index as defined by SSI = (number of employees)^2/county work force.

The problem with weak correlations is that they are easily confounded by socioeconomic relationships between industries and between diseases. These socioeconomic or geographical relationships can produce stronger correlations than true industry-disease correlations. Thus, the problem becomes one of differentiating between a few true correlations and many confounding correlations.

Looking at it in this light, we can define the characteristics of industries that would be expected to produce high industry-disease correlations (if a real effect exists) and, therefore, would be more likely to be differentiated from socioeconomic confounding. Industries that are the sole or primary element of an SIC category will minimize the effect of industrial dilution and therefore provide stronger correlations. Likewise, industries that are generally located in rural counties—where the exposed population is a large fraction of the county population—would minimize the effect of county population dilution and maximize the correlation coefficient. Finally, industries that are very intense and concentrated and tend to group together in large towns or cities will produce a large exposed population which will reduce the dilution problem and provide stronger correlations.

It is extremely interesting to observe that the industries that were selected from the regression analysis for study possessed characteristics which provided the best chance for obtaining large correlation coefficients. All three industries (copper mining, steel manufacturing, and bituminous coal mining) were the sole or principal component of their 4-digit SIC categories. Copper mining and bituminous coal mining were rural industries which minimized the county dilution problem. Steel manufacturing is a highly concentrated industry which provides large exposed populations and therefore reduces the county dilution.

Thus, if true disease associations existed for the copper, steel and coal mining industries, it is entirely reasonable that the multiple regression analysis would identify them. Thus, the fact that the field investigations found some evidence of causal relationships for these industries is compatible with what we would expect, and supports the validity and worth of the multiple regression analysis for limited types and characteristics of industries.

We conclude this discussion with the observation that screening and validation of the results is a very important part of the multiple regression analysis. That is, the true associations do not stand out by themselves; it is through screening and statistical validation tests that the valid associations are identified. For example, to identify the copper, steel, and bituminous correlations, we screened almost 800 correlations and statistically validated 39.

3. *Evaluation of Case Study Methodology*

One of the objectives of the study is to develop methods for investigating relationships between industries and diseases. Specific methodological findings and problems that resulted from the four case studies are discussed here.

a. *Size of Exposed Population*

For two of the case studies, copper smelting and bituminous coal mining, the investigation was hampered by the small size of the exposed populations. For the most part, the small populations were a characteristic of the industry. Both copper smelters and coal preparation plants are generally located in rural areas where only relatively small communities are highly exposed.

The problem with small communities is that there are too few deaths to provide a statistically significant comparison between exposed and control areas. The problem is particularly acute for diseases with low mortality rates. Figure II-2 shows the relationship between exposed population, mortality rate, and relative risk. For a given effect (relative risk) in the exposed population, the figure graphs the age- and sex-specific population required to show a statistically significant difference between the exposed and control populations as a function of the mortality rate. For a mortality rate of 10, a relative risk of 2,

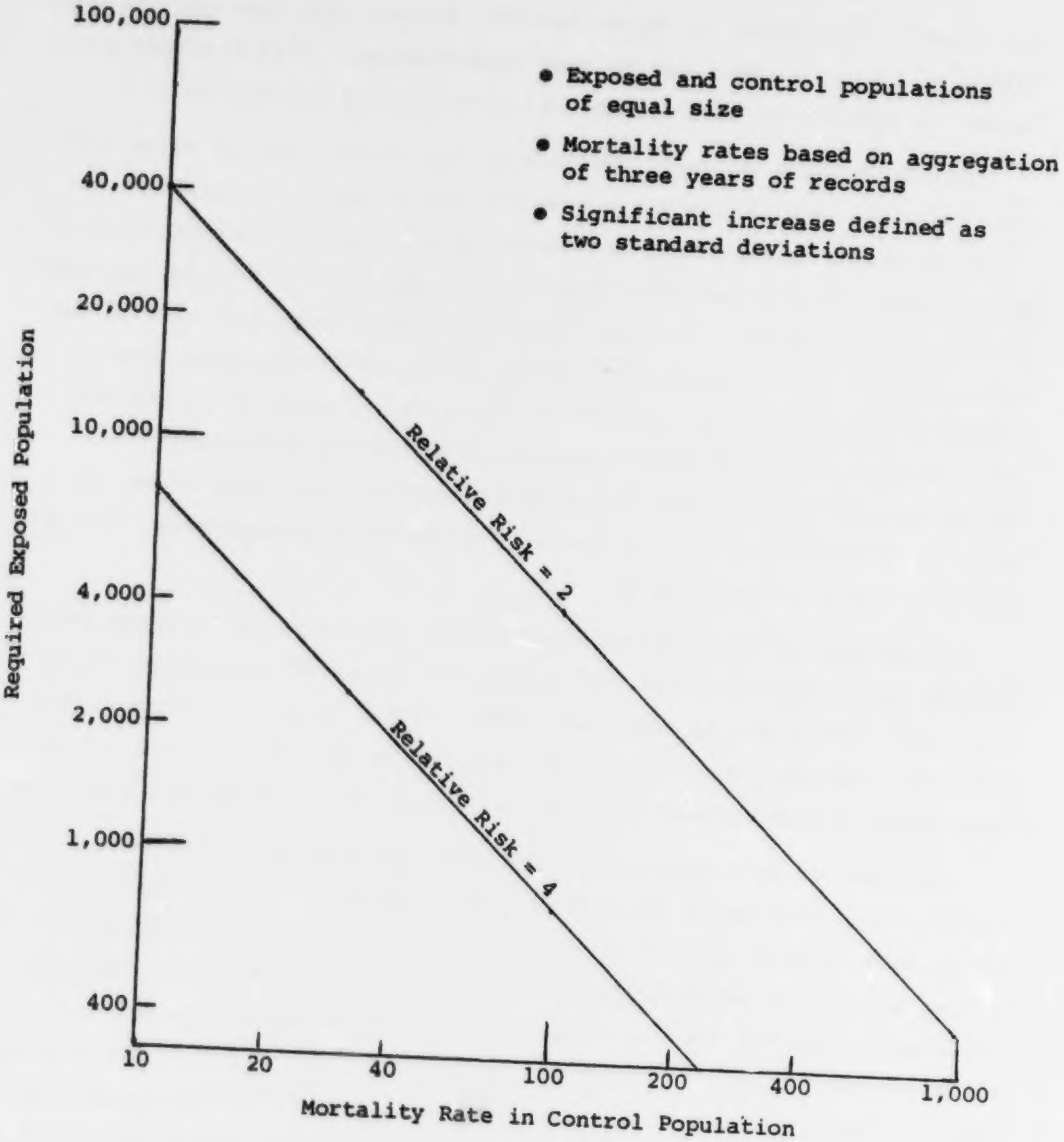


Figure II-2

Magnitude of Exposed Population Necessary
To Show a Statistically Significant Increase in Mortality
for a Given Time and Relative Risk

and 3 years aggregation of death records, an age- and sex-specific population of 40,000 is required to show significance. This required population is reduced to 7,500 if the relative risk is as high as 4.

When restricted to small populations—as in the case of bituminous coal mining—there are only two possible solutions to the problem: (1) aggregate a large number of exposed populations or (2) aggregate a large number of years of data (Figure II-3 shows the effect of the number of years of data). In both cases, certain effects are lost in the aggregation. In the former, it is the effect of differences in site characteristics and plant processes, and in the latter it is the temporal effects. In both the copper smelting and the bituminous coal mining investigations, both types of aggregation were used since the age- and sex-specific populations of individually exposed areas were of the order of a thousand or so.

In the case of steel manufacturing and viscose rayon, sizable populations were available that were relatively clear of industrial clutter. For steel, large cities could have been studied (e.g., Pittsburgh, Birmingham); however, the decision was made early in the study not to select large cities because of urban pollution and socioeconomic problems.

Now that we have experienced the difficulty of obtaining health records over many years (discussed below) and have seen the value of being able to show effects of stratifications, we feel that larger cities with large exposed populations should be investigated. This is especially true for intense, highly concentrated industries such as steel manufacturing. Although multiple sources of pollution are a problem with large cities, examination of health records at the census-tract level should be sufficiently fine-grained to disclose disease patterns associated with the industry. Further, the large exposed populations will enable a significant result to be obtained as a function of time, thus showing temporal trends with industrial activity and control measures.

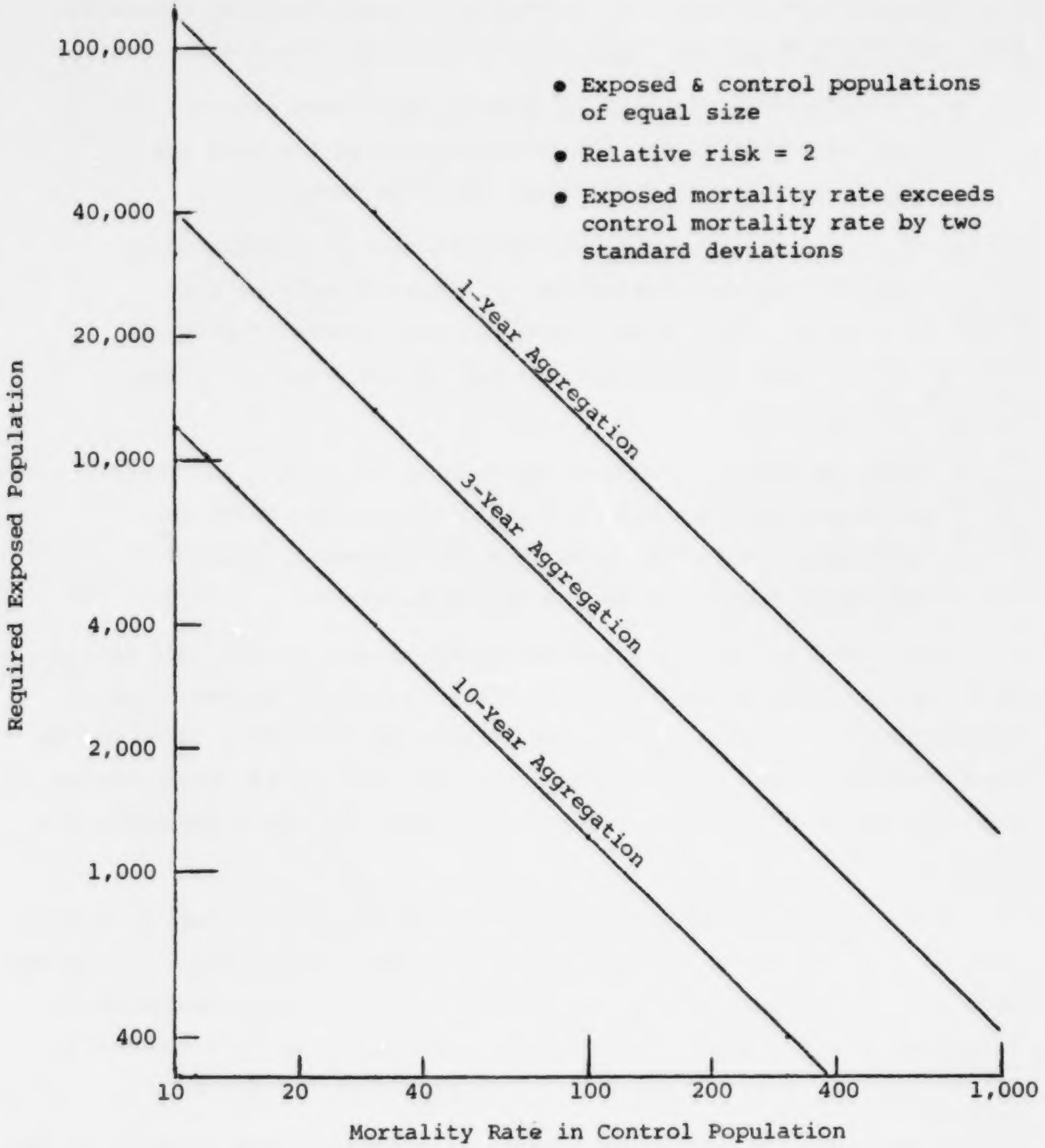


Figure II-3

Effect on Exposed Population Requirements of Aggregating Mortality Records Over Time

b. *Health Data*

Magnetic tapes of death records provide easy, low-cost access to many records; however, we found several problems with them:

- Limited number of years of data on tape (most states started computerizing their records during the last few years, so prior records do not exist on tape).
- Lack of certain information on tape such as occupation or specific address (the latter is a particularly serious problem in urban areas, since the tape generally provides city or town of residence and not census tract or enumeration district).
- They can contain a significant number of errors, and there is no way to check data without going back to death certificates. This had to be done for Johnstown, where errors in town of residence were discovered.

Thus, until states put place of residence such as zip code on tape, hard copies of the death certificates are needed for epidemiological investigations in urban areas. For rural areas, magnetic tapes can be used; however, death certificates are needed for occupational information and for providing data prior to the years for which tape data are available.

It would also be extremely useful for epidemiologic studies if duration of residence were included as well as place of residence. This has been done only recently in a few states. Acquisition of residence information by other means is very time-consuming, requiring on-site inspection of local records such as voter registration, property tax, etc.

We did not find central data banks of morbidity records to be useful in the sites investigated. The cancer registries in Utah did not provide addresses by town or zip code (except for the last three years). Likewise, PAS data for the sites investigated lacked place of residence.

As for health records from hospitals, we found some hospitals eager to help us and some that wanted no part of the study. Hospitals that were associated with the industry being investigated offered to participate if they could be convinced that a health problem existed.

A final note about health data collection—we found, in communicating with vital statistics departments and health registries, that it was a difficult matter to find out precisely what data were available and in what form. Statements would be made about the number of years of information available that would turn out to be incorrect. In several cases, the precise facts about the data were not known until an official request was made for the data. The turnaround time required to fill a request for data was rather variable, ranging from a few weeks to several months.

c. *Control Areas*

Two problems with the matched-control methodology occurred throughout the study:

- Difficulty in finding suitable control populations in the rural areas.
- Lack of air quality measurements for control areas.

The first problem is due to the fact that the industry under investigation affects the socioeconomic characteristics of the area significantly, particularly in rural areas. Hence, towns in the region that do not have a similar industrial base are not similar socioeconomically. In the more urbanized areas, finding suitable control areas is not as difficult.

The second problem constitutes a serious difficulty with the matched-control methodology. EPA and the states are effective at taking air quality measurements in areas containing polluting industries; however, in most areas without pollution sources, few if any measurements are being taken. Thus, it was not possible in most cases to verify that the air quality in the control areas was better than that in the exposed area.

In the present study, this problem could not be solved by taking measurements during the course of the study. In most cases, the industry had incorporated control devices in the past few years; we needed to compare the air quality prior to emission control, since we were studying populations that were exposed prior to emission control. However, in future studies of causal relationships by EPA, the focus should be on the health problems that the industry is causing currently or in the past several years since controls were adopted. Hence, the problem with the lack of air quality measurements in control areas can be taken care of by going out and measuring the air quality.

d. *Exposure Confirmation*

It had been planned to confirm the exposure of decedents by determining the length of residence from a check of state and local records. This was attempted in Wyoming County with tax records and found to be a time-consuming and expensive procedure. For lack of time and resources, it was not carried through. In Arizona, the length of residence in the state was indicated on the death certificate; however, the populations were too small to effectively analyze the mortalities by length of residence. Furthermore, the length of residence in the state was not a good indicator of the exposure (i.e., length of residence in exposed towns).

Any further validation of the industry-disease relationships should confirm and analyze the length of residence. This information would be particularly useful where the exposed populations are sufficiently large to permit examination of temporal effects. To reduce the cost of this effort, a 10% random sample would provide a sufficiently strong indication of the exposure of the total decedents.

e. *Missing, Incomplete, and Incorrect Data*

A troublesome problem with most sites was missing and incomplete data. This was particularly true for water and air quality data which was often spotty. In some cases, the measurements were obviously incorrect. This points out the importance of the site selection effort. Extra effort should be spent in checking the accuracy and completeness of the data available prior to selecting the site.

f. *Smoking Histories*

Other than qualitative information, no data on smoking habits of the mortalities and morbidities were obtained. The matched-control methodology should in general provide a reasonable match of smoking habits, unless the presence of the industry influences the habit—as might be the case with bituminous coal mining. Cigarette consumption data can be developed by county, but probably not by community. Although these data were not developed in this study, it would be useful in further work to assist in the control matching. To fully pursue the influence of smoking on the causal relationships, smoking histories of the decedents in both the control and the exposed populations would be required. This is an extensive effort that should be performed only if maximum "proof" of a relationship is required.

Chapter III

CONCLUSIONS AND RECOMMENDATIONS

A. CONCLUSIONS AND RECOMMENDATIONS PERTAINING TO INDUSTRY-DISEASE RELATIONSHIPS

(1) In three of the four cases studied, substantial evidence was found which indicates that pollutants from the industry in question could be the source of disease in the community. In no case was the evidence sufficient to be conclusive and, in general, represented only a small fraction of the epidemiological evidence needed to establish and confirm cause-and-effect relationships. This was to be expected considering the exploratory nature of the work, principally concerned with investigating methodologies. With these reservations in mind, the following conclusions and observations regarding industry-disease relationships are made:

(a) SO_2 and particulate emissions from copper smelters may be instrumental in causing excessive mortalities from respiratory diseases in the exposed communities.

(b) Abuse of alcohol is the probable cause of excessive digestive diseases in copper smelter communities.

(c) Organic emissions from coking and sintering operations associated with steel manufacturing are the likely cause of excessive mortalities from cancers of the oral, respiratory, digestive, and genitourinary systems in communities exposed to high concentrations of steel plant emissions.

(d) Coal dust emissions from thermal-dryer preparation plants and coal transport vehicles are a possible cause of elevated respiratory and cardiovascular diseases in bituminous coal mining communities.

(e) Emissions or effluents from viscose rayon plants do not appear to cause a significant community health problem.

(2) Further study is required to provide conclusive evidence of the industry-disease relationships identified for the copper smelting, steel, and coal mining industries (items (a), (c), and (d) above). The following studies are recommended:

(a) A continuation of the investigation of the association between copper smelting and respiratory/digestive diseases, to include:

- field investigation of additional sites, in particular, Tacoma, Washington;
- collection and analysis of morbidity records;
- verification of the exposure of mortalities/morbidities by determining length of residence;
- collection and analysis of alcohol consumption data.

(b) A continuation of the investigation of the association between the steel industry and various diseases, to include:

- investigation of additional sites, in particular, Pittsburgh, Buffalo, Detroit, and Birmingham;
- verification of exposure by determining length of residence;
- collection and analysis of hospital and cancer registry records.

(c) A continuation of the investigation of the association between the bituminous coal mining industry and respiratory/cardiovascular diseases, to include:

- investigation of additional sites;
- collection and analysis of additional mortality records for as many years as possible;
- collection and analysis of hospital records;
- air quality measurements to verify differences in coal dust concentrations between exposed and control areas.

B. CONCLUSIONS AND RECOMMENDATIONS PERTAINING TO THE MULTIPLE REGRESSION ANALYSIS

(1) For certain types of industry-disease associations, the results of the study have demonstrated that the multiple regression analysis of county mortality and industry data is effective in identifying associations having a plausible cause-and-effect basis founded on etiological considerations and mortality-pollutant patterns at the local level. The types of industry-disease associations that appear to be identifiable through the multiple regression analysis are those in which (1) the industry is the sole or principal component of the four-digit SIC category and (2) the industry is sufficiently intense or sufficiently rural to expose a large portion of the county population within which it resides. This conclusion is based on the results of the three case studies selected from the multiple regression analysis. Each of the industry-disease associations studied satisfied both of the criteria above, and each of the case studies provided some evidence of possible causal relationships between the industry and associated diseases.

(2) The value of the multiple regression approach for identifying true associations for industries which are not the principal component of the SIC category or which are not sufficiently intense or rural to expose a large portion of the county population is unknown. To explore this capability of the regression approach, further case studies need to be performed.

(3) The results of the study with regard to the effectiveness and value of the multiple regression approach are positive and encouraging. To further develop the capabilities of the approach and to further understand its limitations, the following recommendations are offered:

(a) Continue the field studies of copper smelting, steel manufacturing, and bituminous coal mining to provide stronger evidence that true relationships exist and, thereby, provide confirmation that the multiple regression approach is effective. (See Section A(2) above for specific recommendations for these studies.)

(b) Investigate other industry-disease correlations identified by the multiple regression analysis. The characteristics of the industries selected for these new case studies should vary with regard to SIC category dominance and capability for exposing large fractions of the county population. In this way, these studies will provide a means for assessing the limitations of the regression approach.

(c) Perform a study to develop an industrial index that will be a better estimator of dosage to exposed communities. The study should examine (1) the value and feasibility of developing different indices for different classes of industries; (2) the feasibility of incorporating age of industry to provide a measure of duration of exposure; and (3) the feasibility of utilizing indices that correlate closely with point source emission estimates for a given class of industry.

(d) With the improved industrial index (see (c) above), rerun the multiple regression analysis with recent mortality data (1972-1977) to identify industry-disease correlations more in line with current waste control policies and procedures.

C. CONCLUSIONS AND RECOMMENDATIONS PERTAINING TO METHODOLOGY FOR INVESTIGATING INDUSTRY-DISEASE RELATIONSHIPS

The basic methodology used for investigating industry-disease relationships consisted of a matched-control approach in which health and environmental data are compared for exposed and matched-control areas to identify diseases whose incidence or prevalence can be associated with emissions or effluents produced by the industry. Based on the results of the four case studies, certain conclusions and recommendations can be made regarding this methodology.

(a) The investigation of multiple sites is critical to establishing and confirming true relationships. Variation in effect of industry and difficulty in finding matched-control populations make multiple site investigations a necessity. Two sites were found to be insufficient to provide satisfactory evidence.

(b) Sites with small populations should be avoided, even though they are ideal from other points of view (e.g., lack of industrial clutter and other sources of pollution). The difficulty in gathering health data for a significant number of years and the weakening of the analysis due to inability to examine stratifications of the data make these sites relatively ineffective for investigation of causal relationships. Although large city sites were not studied, it is believed that the advantages of large exposed populations would outweigh the disadvantages; i.e., the added strength of analysis associated with large exposed populations would more than compensate for the problems with industrial clutter and urban pollution.

(c) A weakness in the matched-control methodology is the general unavailability of good air quality data for the control areas. Taking measurements of the current air quality is not necessarily the answer, since these measurements do not indicate the air quality during prior years.

(d) Effective use of time trends could not be made, due to the need for aggregating the health data over time and due to the difficulty of obtaining information on exposure duration (length of residence) for the affected individuals. Hence, no conclusions can be made about the value of temporal trends for investigating industry-disease relationships.

(e) Neither centralized morbidity data systems nor records acquired from hospitals could be used effectively. The former generally lacked needed place-of-residence information, and the latter could not be obtained for a meaningful percentage of the hospitals in the study areas. Based on our findings, we conclude that putting the effort into investigation of the mortalities at additional sites is more valuable and has a greater chance of success than putting the same amount of effort into collecting and analyzing hospital morbidity records for a given site. When centralized sources of morbidity data include adequate address information, morbidity records should prove useful in the investigation of causal relationships. It is recommended that steps be taken by EPA to encourage state and local disease registries to include census tract or town of residence information on their morbidity records.

(f) Interpretation of aerial photography for previous years was useful in providing historical evidence of changes in population and pollution point sources (i.e., stacks, control devices, or effluent discharges). However, for the most part, this information was more readily available from other sources to the accuracy required in the study. Based on this experience, we feel that retrospective photointerpretation is valuable only when corroborative evidence is required or in special cases where the information is not available elsewhere. Although not studied here, we feel that photointerpretation would have greater value in present-day or prospective studies, where the data can be collected as desired and use can be made of various remote sensing techniques to provide more quantitative information on the pathways and the dispersion patterns of emissions and effluents.

Chapter IV

STUDY METHODOLOGY

A. *OVERVIEW*

This chapter discusses the various methods and procedures used in all phases of this study. The initial phase of the study, performed under a separate EPA contract, identified correlations between broad industry groups (as defined by Standard Industrial Classification [SIC] codes) and categories of disease mortality. These correlations were then screened and validated to choose those most significant and best suited for further investigation.

Following this, study sites were selected and hypotheses were developed and tested in separate sites. Data on mortality and morbidity, air and water quality, etiology, and industry characteristics were collected, analyzed, and integrated to arrive at the conclusions presented in this report.

While each industry-disease correlation selected for study was investigated using a somewhat different protocol, the general methodology was essentially the same. This general methodology and the specific procedures comprising it are discussed in this chapter.

B. *INITIAL CORRELATIONS*

The initial industry-disease correlations were generated primarily by a multiple regression analysis of disease-specific, age-adjusted mortality rates by county (for years 1968-1972) on indices of industrial activity for some 460 industries, based on 1959 data. These industries were, in some cases, actually groups of industries (sometimes unrelated with respect to type or process) which were consistently located in the same counties. The index of industrial activity was a function of the number of employees in the industry and the total work force in the county. This analysis was done separately for males and females and for whites and nonwhites. The nonwhite regression had some calculation errors which could not be corrected in time for inclusion in the later study phases, so the investigation considered only the associations between industrial activity and mortality in whites.

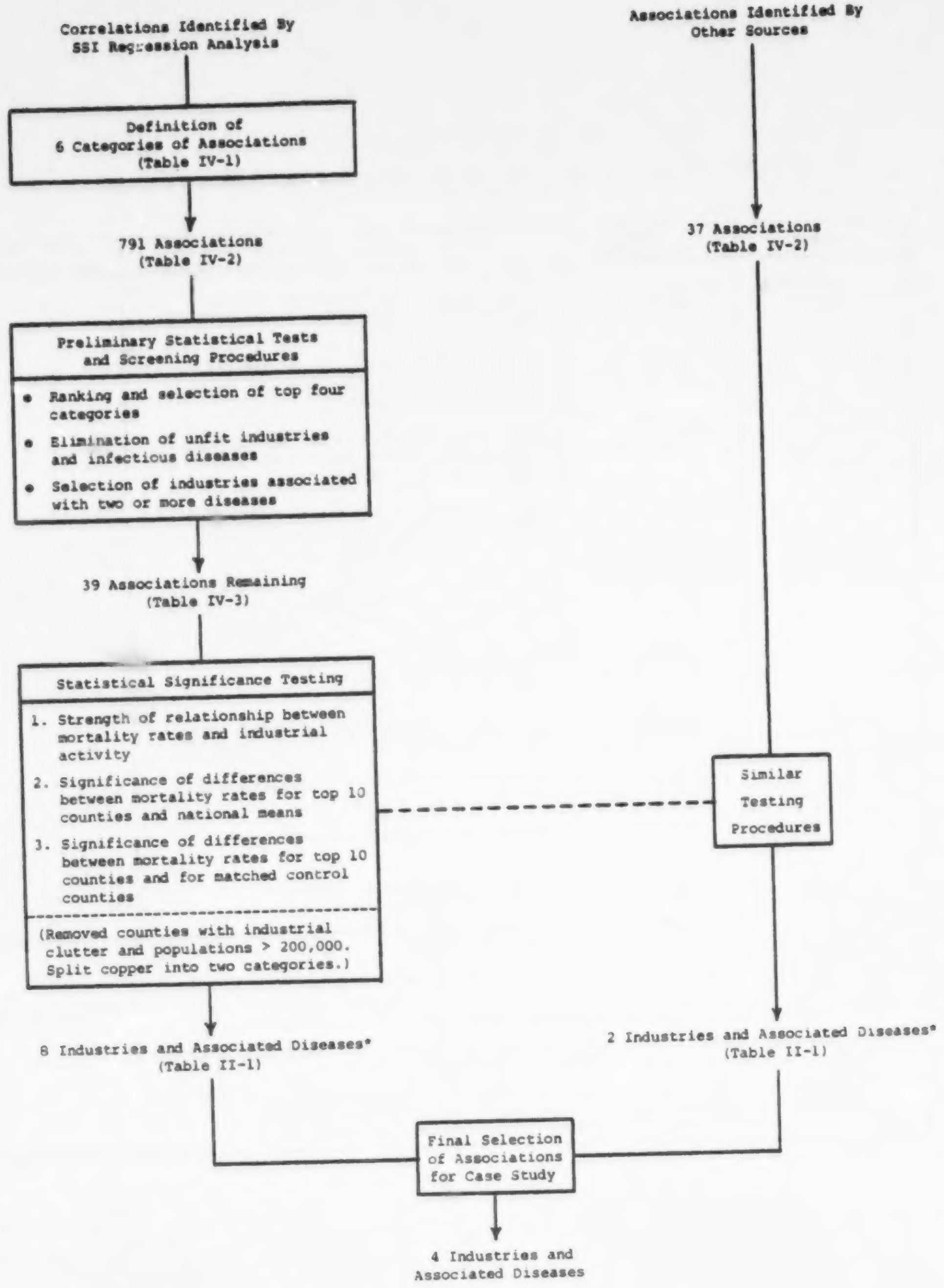
In addition, the counties were divided by EPA randomly into two groups for the regression analysis. Therefore, the separate regressions for each half of the total counties and for each sex provided a measure of the consistency of the derived correlations. However, even a strong association might not be reflected as correlations in all subsamples since males are more likely to be exposed occupationally, and either subset of counties might not necessarily contain a particular industry.

A total of 791 industry-disease correlations resulted from the regression approach. Other sources of potential associations were used to augment those found through the regression analysis and to provide a means for the screening procedures to determine the strongest testable associations regardless of source. The multiple regressions constituted a completely objective source providing the largest number of possible associations. The other potential associations were generated by manual inspection of county industry and mortality data, through literature searches and from observations made by State health officials on environmental health problems. Thirty-six associations were identified in this manner.

C. *SCREENING AND VALIDATION*

Correlations from the regression analysis were ranked and then grouped into six main categories containing 791 associations. These categories were based on the values of R and Δr^2 in the regression and whether or not the derived correlations were present in both subsamples of counties and in males and females. Table IV-1 describes the six categories and Table IV-2 lists the correlations by category. Also listed in Table IV-2 are the 37 associations identified by means other than the regression analysis.

By a series of statistical tests and screening procedures outlined in Figure IV-1, a small number of the strongest correlations were selected from the 791 associations identified by the regression analysis. These served as the candidate associations for field study. The procedure by which the 791 associations were boiled down to a few is described below.


Correlations Identified By SSI Regression Analysis
Associations Identified By Other Sources
Definition of 6 Categories of Associations (Table IV-1)
791 Associations (Table IV-2)
37 Associations (Table IV-2)
Preliminary Statistical Tests and Screening Procedures
Ranking and selection of top four categories
Elimination of unfit industries and infectious diseases
Selection of industries associated with two or more diseases
39 Associations Remaining (Table IV-3)
Statistical Significance Testing
1. Strength of relationship between mortality rates and industrial activity
2. Significance of differences between mortality rates for top 10 counties and national means
3. Significance of differences between mortality rates for top 10 counties and for matched control counties
(Removed counties with industrial clutter and populations > 200,000. Split copper into two categories.)
Similar Testing Procedures
8 Industries and Associated Diseases* (Table II-1)
2 Industries and Associated Diseases* (Table II-1)
Final Selection of Associations for Case Study
4 Industries and Associated Diseases

*Candidate associations for field study.

Figure IV-1. Procedures for Screening and Selecting Industry-Disease Associations for Case Study

Table IV-1

SCREENING CRITERIA FOR INDUSTRY-DISEASE ASSOCIATIONS

SOURCE	GROUP DESIGNATION	SCREENING CRITERIA	NUMBER OF ASSOCIATIONS
I. Regression Data[a]	a) Upper MF-AB	Correlation in both males and females Correlation in both sample A and sample B Upper R and Δr^2 class[b]	25
	b) Lower MF-AB	Correlation in both males and females Correlation in both sample A and sample B Lower R and Δr^2 class[c]	31
	c) Upper M or F-AB	Correlation in either males or females Correlation in both sample A and sample B Upper R and Δr^2 class[b]	15
	d) Lower M or F-AB	Correlation in either males or females Correlation in both sample A and sample B Lower R and Δr^2 class[c,d]	548
	e) Upper MF-A or B	Correlation in both males and females Correlation in either sample A or sample B Upper R and Δr^2 class[b]	16
	f) Upper M or F-A or B	Correlation in either males or females Correlation in either sample A or sample B Upper R and Δr^2 class[b]	156 (791)
II. Data Inspection	Inferred Associations	Association between high industrial activity and excess mortality	24
III. Basic Principles	Basic Associations	Industrial processing of known disease-causing agents	5
IV. Literature	Recorded Associations	Recorded occupational associations	7
V. Health Officials	Field Associations	Problems identified by officials Supportive epidemiological evidence	1 (37)

[a] All data is uncorrected, untruncated, weighted, and white.

[b] Upper R and Δr^2 class ($R > 0.5$ and $\Delta r^2 > 0.01$ in at least one subsample of males and females).

[c] Lower R and Δr^2 class (residue from upper MF-AB, i.e., $R < 0.5$ or $\Delta r^2 < 0.01$).

[d] 260 potential associations for males and 288 for females were identified in this category. The listing is not included in this report.

Nomenclature: M = male; F = female; A = sample A of U.S. counties; B = sample B of U.S. Counties; R = multiple correlation coefficient; Δr = change in correlation coefficient

Table IV-2. POTENTIAL INDUSTRY-DISEASE ASSOCIATIONS

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATION	
		DISEASE[a]	INDUSTRY
I. Regression Data	a) Upper MF-AB	06 - Neoplasm, digestive organs & peritoneum	P030 - Jewelry, toys, wire drawing
		08 - Neoplasm, small & large intestines	P030 - Jewelry, toys, wire drawing
		09 - Neoplasm, rectum	P030 - Jewelry, toys, wire drawing
		27 - Major cardiovascular disease	2321 - Men's dress shirts & nightwear 121R - Bituminous mining
		28 - Rheumatic heart disease	4932 - Gas & other services combined
		29 - Hypertensive disease	34RR - Fabricated metal products
		32 - Chronic ischemic heart disease & angina pectoris	P001 - 55 big city industries 41RR - Local passenger transportation P052 - River transportation & petroleum
		38 - Diseases of the respiratory system	102R - Copper mining
		39 - Acute interstitial & broncho-pneumonia	316R - Luggage P001 - 55 big city industries
		40 - Bronchitis, emphysema & asthma	102R - Copper mining
		42 - Emphysema	102R - Copper mining
		45 - Other diseases of the respiratory system	121R - Bituminous mining
		46 - Diseases of the digestive system	41RR - Local passenger transportation P001 - 55 big city industries
		47 - Cirrhosis of liver	P001 - 55 big city industries
		48 - Alcoholic cirrhosis of liver	P018 - Aircraft P001 - 55 big city industries 2084 - Wines, brandy & brandy spirits
		49 - Other & unspecified cirrhosis of liver	41RR - Local passenger transportation 3273 - Ready mixed concrete P041 - Transportation
	b) Lower MF-AB	08 - Neoplasm, small & large intestines	P036 - Motors & generators
		11 - Neoplasm, esophagus, liver, gallbladder, peritoneum & other digestive organs	P001 - 55 big city industries
		27 - Major cardiovascular disease	P052 - River transportation & petroleum
		29 - Hypertensive disease	P061 - Footwear
		30 - Ischemic heart disease	2321 - Men's dress shirts & nightwear 121R - Bituminous mining P052 - River transportation & petroleum P047 - Steel 32PB - Glass
		31 - Acute myocardial infarction	121R - Bituminous mining P061 - Footwear 2321 - Men's dress shirts & nightwear
		32 - Chronic ischemic heart disease & angina pectoris	121R - Bituminous mining 327R - Concrete, gypsum & plaster
		34 - Cerebrovascular disease	P069 - Phonograph records & transmissions
		38 - Diseases of the respiratory system	121R - Bituminous mining

(continued)

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[a] These are disease codes for 56 disease categories used by SSI in the regression analysis (see Appendix J).

Table IV-2 (continued)

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATION	
		DISEASE	INDUSTRY
I. Regression Data (cont.)	b) Lower MF-AB (cont.)	39 - Acute interstitial & broncho-pneumonia	121R - Bituminous mining
		40 - Bronchitis, emphysema & asthma	121R - Bituminous mining
		41 - Bronchitis	121R - Bituminous mining
		42 - Emphysema	P006 - Transportation, aircraft, pottery, etc.
		46 - Diseases of the digestive system	102R - Copper mining 2951 - Paving mixtures & blocks 2084 - Wines, brandy & brandy spirits
		47 - Cirrhosis of liver	41RR - Local passenger transportation 2084 - Wines, brandy & brandy spirits 102R - Copper mining
		48 - Alcoholic cirrhosis of liver	102R - Copper mining 3273 - Ready mixed concrete
		49 - Other & unspecified cirrhosis of liver	P001 - 55 big city industries 2291 - Felt goods, N.E.C. 327R - Concrete, gypsum & plaster
	c) Upper M or F-AB		
	Males	06 - Neoplasm, digestive organs & peritoneum	P047 - Steel 415R - School buses
		11 - Neoplasm, esophagus, liver, gallbladder, peritoneum & other digestive organs	P003 - Foods, machinery & communications
		13 - Neoplasm, trachea, bronchus & lung	P052 - River transportation & petroleum 209R - Miscellaneous food preparations
		38 - Diseases of the respiratory system	P059 - Cigars & anthracite mining
		48 - Alcoholic cirrhosis of liver	316R - Luggage
	Females	15 - Neoplasm, breast	P001 - 55 big city industries
		27 - Major cardiovascular disease	P047 - Steel
		28 - Rheumatic heart disease	279R - Printing trades services
		31 - Acute myocardial infarction	P047 - Steel
		49 - Other & unspecified cirrhosis of liver	P006 - Transportation, aircraft, pottery, etc.
		NOTE: In the following correlations, $\Delta r^2 > 0.01$, but $R < 0.5$.	
	Males	55 - Diseases of the skin & subcutaneous tissue	4171 - Bus terminal facilities
	Females	16 - Neoplasm, female genital organs	P003 - Foods, machinery & communications
		37 - Diseases of veins, lymphatics & other diseases of circulatory system	121R - Bituminous mining
	d) Lower M or F-AB	(548 potential associations were identified in this category. Because of the large number and the weakness of the correlations, the listing is not included here.)	

(continued)

Table IV-2 (continued)

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATION	
		DISEASE	INDUSTRY
I. Regression Data (cont.)	e) Upper MF-A or B	08 - Neoplasm, small & large intestines	PO30 - Jewelry, toys, wire drawing 2649 - Paper & paper board products
		27 - Major cardiovascular disease	PO30 - Jewelry, toys, wire drawing
		31 - Acute myocardial infarction or other acute ischemic heart disease	259R - Misc. furniture & fixtures 102R - Copper mining 3273 - Ready mixed concrete
		32 - Chronic ischemic heart disease & angina pectoris	279R - Printing trade services
		38 - Diseases of the respiratory system	3421 - Cutlery 102R - Copper mining
		39 - Diseases of the digestive system	327R - Concrete, gypsum & plaster
		47 - Cirrhosis of liver	327R - Concrete, gypsum & plaster
		48 - Alcoholic cirrhosis of liver	PO38 - Motors, tool & die, metal industries 259R - Misc. furniture & fixtures 2084 - Wines, brandy & brandy spirits
		49 - Unspecified cirrhosis of liver	3421 - Cutlery 3273 - Ready mixed concrete
	f) Upper A or B Males	05 - Neoplasm, buccal cavity & pharynx	3672 - Cathode ray picture tubes PO42 - Ind. gases, syn. rubber, cooperage 2591 - Venetian blinds & shades 349R - Fabricated metal products 279R - Printing trades services 2951 - Paving mixtures & blocks PO18 - Aircraft 2393 - Textile bags
		06 - Neoplasm, digestive organs	3672 - Cathode ray picture tubes 279R - Printing trades services PO47 - Steel 3421 - Cutlery PO30 - Jewelry, toys, wire drawing 349R - Fabricated metal products PO27 - Asphalt, asbestos, coal tar 3623 - Welding apparatus PO47 - Steel 2951 - Paving mixtures & blocks
		07 - Neoplasm, stomach	PO30 - Jewelry, toys, wire drawing 38PA - Optics PO61 - Footwear, tanning, lamps, misc. textile PO47 - Steel
		08 - Neoplasm, small & large intestines	3672 - Cathode ray picture tubes 3297 - Non-clay refractories PO30 - Jewelry, toys, wire drawing PO27 - Asphalt, gaskets, etc. 3623 - Welding apparatus 349R - Fabricated metal products 3841 - Surgical & medical instruments 2032 - Canned specialties
		09 - Neoplasm, rectum	279R - Printing trades services PO47 - Steel 3334 - Primary aluminum 372R - Aircraft & parts PO30 - Jewelry, toys, wire drawing 3554 - Paper industries machinery 349R - Fabricated metal products 2295 - Coated fabric, non-rubber 354Q - Machine tools, metal working machinery 3251 - Brick & struct. clay tile

(continued)

Table IV-2 (continued)

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATION	
		DISEASE	INDUSTRY
I. Regression Data (cont.)	f) Upper A or B Males (cont.)	11 - Neoplasm, esophagus, liver, gallbladder, peritoneum & other digestive organs	3672 - Cathode ray picture tubes 349R - Fabricated metal products 2951 - Paving mixtures & blocks
		13 - Neoplasm, trachea, bronchus & lung	209R - Miscellaneous food preparations PO42 - Ind. gases, syn. rubber, etc. 2591 - Venetian blinds & shades 36PO - Batteries, wiring devices 121R - Bituminous mining 2295 - Coated fabric, non-rubber 295R - Paving & roofing materials 202R - Dairy products 2951 - Paving mixtures & blocks
		14 - Neoplasm, other respiratory organs	3672 - Cathode ray picture tubes 2591 - Paving mixtures & blocks PO61 - Footwear, tanning, etc. 349R - Fabricated metal products
		24 - Diabetes mellitus	PO38 - Motors, etc., metals industries 2295 - Coated fabric, non-rubber 366P - Communication equipment 2591 - Venetian blinds & shades 2821 - Plastics materials PO30 - Jewelry, toys, wire drawing 3623 - Welding apparatus 3273 - Ready mixed concrete 36PC - Electrical equipment
		27 - Major cardiovascular disease	3273 - Ready mixed concrete 259R - Misc. furniture & fixtures 355R - Special ind. machinery 22PC - Rugs, synthetic weaving 349R - Fabricated metal products
		31 - Acute myocardial infarction	PO43 - Semiconductors, tubes, scientific instruments 259R - Misc. furniture & fixtures 38PB - Mechanical instruments, etc. 3822 - Automatic temperature controls 3273 - Ready mixed concrete 3631 - Household cooking equipment 2282 - Yarn throwing & winding & mills
		32 - Chronic ischemic heart disease & angina pectoris	36PD - Batteries, wiring devices 2295 - Coated fabric, rubberized 3672 - Cathode ray picture tubes 36PB - Elec. meas. instru., etc. 366P - Communications equipment 2951 - Paving mixtures & blocks
		33 - Other heart disease	3672 - Cathode ray picture tubes 2042 - Prepared animal feed 2091 - Cotton seed oil mills 261R - Pulp mills 3662 - Radio, tv communication equip.
		34 - Cerebrovascular disease	314R - Footwear 2393 - Textile bags 361P - Transformers 22PB - Weaving mills, cotton 366P - Communication equipment PO30 - Jewelry, toys, wire drawing 22PC - Carpets, weaving, synthetics 3273 - Ready mixed concrete
		35 - Arteriosclerosis	2299 - Textiles 2951 - Paving mixtures & blocks 3672 - Cathode ray picture tubes
		38 - Diseases of respiratory system	121R - Bituminous mining 316R - Luggage 333R - Nonferrous metals 3333 - Zinc

(continued)

Table IV-2 (continued)

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATION	
		DISEASE	INDUSTRY
I. Regression Data (cont.)	f) Upper A or B Males (cont.)	47 - Cirrhosis of liver	314R - Footwear, except rubber PO17 - Residuals in metal ores, etc. PO18 - Aircraft engines & props 2951 - Paving mixtures & blocks
		48 - Alcoholic cirrhosis of liver	PO18 - Aircraft engines & props 38PA - Optics 314R - Footwear, except rubber 336R - Nonferrous foundries 3672 - Cathode ray picture tubes 3421 - Cutlery 2063 - Beet sugar 316R - Luggage
		49 - Other & unspecified cirrhosis of liver	3591 - Venetian blinds & shades 279R - Printing trades services PO38 - Machines, hardware, etc. PO47 - Steel PO30 - Jewelry, toys, wire drawing 2951 - Paving mixtures & blocks
		50 - Other digestive diseases	3822 - Mechanical measuring devices
	Females	08 - Neoplasm, small & large intes.	279R - Printing trades services
		12 - Neoplasm, respiratory tract	3273 - Ready mixed concrete 3672 - Cathode ray picture tubes
		13 - Neoplasm, trachea, etc.	3273 - Ready mixed concrete
		27 - Major cardiovascular disease	PO47 - Steel 3442 - Metal doors, sash & trim 2649 - Paper & paper board products
		28 - Chronic rheumatic heart disease	279R - Printing trades services 3421 - Cutlery PO30 - Jewelry, toys, wire drawing
		29 - Hypertensive disease	3423 - Edge tools PO30 - Jewelry, toys, wire drawing 3672 - Cathode ray picture tubes 2295 - Coated fabric, non-rubber
		30 - Ischemic heart disease	PO47 - Steel 3442 - Metal doors, sash & trim PO38 - Motors, etc., metals industries PO30 - Jewelry, toys, wire drawing 2649 - Paper & paper board products
		31 - Acute myocardial infarction & other acute ischemic heart disease	4932 - Gas & other services combined PO47 - Steel 3442 - Metal doors, sash & trim PO38 - Motors, etc., metals industries 2649 - Paper & paper board products
		32 - Chronic ischemic heart disease	4932 - Gas & other services combined
		39 - Acute interstitial & broncho-pneumonia	279R - Printing trades services 341R - Fabricated metal parts PO30 - Jewelry, toys, wire drawing 259R - Misc. furniture & fixtures
		47 - Cirrhosis of liver	2084 - Wines, brandy & brandy spirits 3672 - Cathode ray picture tubes
		48 - Alcoholic cirrhosis of liver	279R - Printing trades services 327R - Concrete, plastur & gypsum 102R - Copper mining

(continued)

Table IV-2 (continued)

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATION	
		DISEASE	INDUSTRY
II. Data Inspection	Inferred Associations	05 - Neoplasm, buccal cavity	2421 - Sawmills
		06 - Neoplasm, digestive organs	2421 - Sawmills 2649 - Paper & paper board products 241R - Logging
		18 - Neoplasm, bladder	2327 - Men's trousers 3442 - Metal doors, sash & trim 2394 - Canvas 2421 - Sawmills
		21 - Leukemia	2421 - Sawmills
		24 - Diabetes mellitus	2421 - Sawmills
		31 - Myocardial infarction	2421 - Sawmills 121R - Bituminous mining
		32 - Other heart disease	121R - Bituminous mining 2321 - Men's dress shirts & nightwear 2341 - Clothing 2328 - Men's clothing
		35 - Arteriosclerosis	22-- - Textiles 31-- - Leather
		39 - Pneumonia	2872 - Fertilizers, mixing
		40 - Bronchitis, emphysema, asthma	2421 - Sawmills
		41 - Bronchitis	2421 - Sawmills 2327 - Trousers 2328 - Men's clothing
		52 - Nephritis/nephrosis	241R - Logging
III. Basic Principles	Basic Associations	Systemic poisoning; respiratory & skin diseases	Pesticide & herbicide use & manufacturing (highly toxic, long-lasting agents which can enter through several bodily routes).
		Congenital malformations	Pesticides, industrial processes involving molybdenum, manganese, zinc, mercury & lead. (Since the developing fetus is susceptible to external agents, a study of congenital malformations in industrialized areas may reveal new interactions of environmental teratogens.)
		Mesothelioma; asbestosis	Production of asbestos. (Highly toxic in small doses; one exposure is often sufficient to produce disease. Minute fibers can linger in air and lungs for long periods. Contamination of communities near asbestos production is likely.)
		Respiratory disease	Smelters and foundries
		Leukemia	Cathode ray picture tubes; nuclear power plants

(continued)

Table IV-2 (concluded)

SOURCE	GROUP DESIGNATION	POTENTIAL ASSOCIATIONS	
		DISEASE	INDUSTRY
IV. Literature	Recorded Associations	Neoplasm, respiratory tract;	Petrochemicals, sulfur, sulfuric acid, synthetic rubber, liquid hydrocarbons, magnesium, fertilizers (Houston Ship Channel Area).*
		*Citation: MacDonald, Preprint (1976)	
		Neoplasm, bladder, lung & liver in males	Chemical industry, i.e., dyes, cosmetics, pharmaceuticals, soaps, rubber (139 industrialized counties).*
		*Citation: Hoover and Fraumeni, Environ. Res. 9:196 (1975)	
		Neoplasm, nasal & bladder	Leather finishing.*
		*Citation: Acheson, Brit. Med. J. 1:385 (1970) Cole, Brit. J. Ind. Med. 11 (1954)	
		Liver angiosarcoma	Vinyl chloride.*
		*Citation: Creech, J. Occup. Med. 16:150 (1974)	
		Neoplasm, lung in males	Paper, chemical, petroleum & transportation.*
		*Citation: Blot, Am. J. Epidemiol. 103(6):539 (1976)	
		Neoplasm, lung	Uranium mining.*
		*Citation: Cassarett, Toxicology, MacMillan (1975)	
		Cardiovascular diseases	Viscose rayon production.*
		*Citation: Schilling, Am. Heart J. 80:1 (1970)	
V. Health & Pollution Officials	Field Associations	Cardiovascular disease	Viscose rayon products. (A site in Warren County, Virginia, with an excess cardiovascular mortality and heavy viscose rayon production has been identified. Preliminary profiles of the industrial process and popualtion at risk have been compiled.)

The six categories of regression data were ranked independently by seven of the ECI staff on the basis of the potential of the various groups to distinguish viable correlations. The top four categories determined by consensus were (a), (b), (c), and (e) which contained 87 associations.

From this group of 87 associations we eliminated industry categories obviously unfit for the study, such as non-point-source industries (transportation) and large conglomerate industry groupings (55 big city industries). Because of its infectious etiology, rheumatic heart disease was eliminated. Fifty-seven associations remained after this process.

We then selected the 39 associations in which industries were associated with two or more similar diseases. This does not imply that nonspecificity was used as a criterion for selecting associations. Rather, it aimed at industries which appeared to exert their effects on several sites within an organ system. These associations are listed in Table IV-3.

The remaining associations were then examined to determine which were statistically significant, unconfounded by nonindustrial variables, and likely to be related causally. For this we used a step-wise process involving three sequential tests.

The first test consisted of determining the statistical significance of mortality rates for the counties with high industrial activity, and then examining the correlation between the degree of significance and the industrial activity. The second test consisted of aggregating the mortality rates for the top 10 or so counties with the highest amount of the industry in question, and testing the statistical significance between the aggregated mortality rates and the national mean mortality rates for the associated diseases. The third test consisted of a mortality control test, in which the aggregated mortality rates for the top 10 counties were compared to the aggregated mortality rates for a set of control counties, which did not contain the industry in question but whose socioeconomic characteristics matched those of the industrial counties.

The last test was by far the most meaningful test, but involved considerably more effort than the others and, therefore, was applied only to those associations that survived the first two tests.

Table IV-3

SCREENED INDUSTRY-DISEASE ASSOCIATIONS FROM REGRESSION DATA ASSOCIATED WITH MORE THAN ONE SIMILAR DISEASE

SIC #	INDUSTRY	DISEASE CATEGORY	SPECIFICS
327R	Concrete, gypsum & plaster	Cardiovascular diseases	32 - Chronic ischemic heart disease (Ia)[a]
		Digestive diseases	47 - Cirrhosis (Ie) 49 - Other cirrhosis (Ib) 46 - Diseases of digestive system (Ie)
3273	Ready mixed concrete	Cardiovascular diseases	31 - Myocardial infarction (Ie)
		Digestive diseases	48 - Alcoholic cirrhosis (Ib) 49 - Other cirrhosis
2951	Paving mixtures & blocks	Digestive diseases	46 - Diseases of digestive system (Ib)
PO30	Jewelry, toys, wire drawing	Cardiovascular diseases	27 - Major cardiovascular diseases (Ie)
		Digestive diseases	06 - Neoplasm, digestive system (Ia) 08 - Neoplasm, small & large intestines (Ia,Ie) 09 - Neoplasm, rectum (Ia)
102R	Copper mining	Respiratory diseases	38 - Diseases of respiratory system (Id) 40 - Bronchitis, emphysema, asthma (Ie) 42 - Emphysema (Ia)
		Cardiovascular diseases	31 - Myocardial infarction (Ie)
		Digestive diseases	47 - Cirrhosis (Ib) 48 - Alcoholic cirrhosis (Ib) 46 - Diseases of digestive system (Ib)
1211	Bituminous coal mining	Respiratory diseases	38 - Diseases of respiratory system (Ia) 39 - Acute interstitial & bronchopneumonia (Ib) 40 - Bronchitis, emphysema, asthma (Ib) 41 - Bronchitis (Ib) 43 - Other respiratory diseases (Ia)
		Cardiovascular diseases	27 - Major cardiovascular diseases (Ia) 30 - Ischemic heart disease (Ia) 31 - Myocardial infarction (Ia) 32 - Chronic ischemic heart disease (Ia) 37 - Other circulatory diseases (Ic, females)
[illegible]	Steel	Cardiovascular diseases	27 - Major cardiovascular diseases (Ic, females) 30 - Ischemic heart disease (Ia) 31 - Myocardial infarction (Ic, females)
		Digestive diseases	06 - Neoplasm, digestive system (Ib)
2321	Men's shirts & nightwear	Cardiovascular diseases	27 - Major cardiovascular diseases (Ia) 30 - Ischemic heart disease (Ia) 31 - Myocardial infarction (Ib)
2084	Wines, brandy & brandy spirits	Digestive diseases	46 - Diseases of digestive system (Ib) 47 - Cirrhosis (Ib) 48 - Alcoholic cirrhosis (Ia)

[a] Letters in parentheses refer to Table IV-2 groupings.

In all three tests, a major concern was the effect of county population on the statistical significance of mortality rate differences. The race- and sex-specific populations of the counties used in the regression analysis varied over several orders of magnitude, from less than one hundred for rural counties to over a million for large city counties. Obviously, a high mortality rate for a population of 100 is much less significant than the same mortality rate for a much larger population. The basic statistical approach used to test the significance between mortality rates for different size populations was used for all three screening and validation methods. It is statistically and mathematically equivalent to the chi-square test of the difference between two proportions or percentages.

The first screening technique used was graphical display to test the strength of the relationship between mortality rate and industrial activity. Plots were made for a number of the associations using several different measures of industrial activity. No strong correlations appeared, although some associations showed trends in a positive direction. The cases which showed promising trends eventually were validated by another screening procedure, but the graphical method by itself was not a suitable measure of the strength of association.

For the next screen, sample counties were chosen by taking the 10 with the highest industrial index, [(number of employees)^2/total county work force] for a particular industry, among those with populations less than 200,000.* The population cutoff was made in an attempt to remove counties where there would be extensive industrial activity and to avoid the complications in disease etiology introduced by urban living. Mean age-adjusted mortality rates were calculated. Those rates which were at least two standard deviations above the national mean remained as indicators of potential investigative associations.

Two categories were removed without calculating aggregated rates because of the nature of the industry. [illegible] appeared in [illegible] counties however, only one county had a population less than 200,000, and the association was more likely with urbanization than with printing. A miscellaneous furniture category was also omitted because all 17 counties had populations above the cutoff. The industrial index gave an indication of this distribution: all printing counties had indices less than 0.3, and all furniture counties fell below 0.1.

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*In some instances, there were not 10 counties having populations less than 200,000, and only the counties that did were included.

The remaining industry-disease associations involved 12 industries. The counties included in the aggregates were then examined for complicating industrial activity. Eliminated from the aggregate were counties with mining, smelting, or chemical industries, as well as those with any industry with an SIC code having the same first two digits as the industry of interest. As a result, the industry grouping "anthracite coal mining and cigars" (P059) was eliminated because eight of the ten sample counties contained other industries considered to be potential complications, i.e., inorganic chemical manufacture, blast furnaces, and bituminous coal. Removal of those eight counties lowered the number of standard deviations above the U.S. mean from 33.0 to 0.1. It was also noted that the industry being examined was mainly cigars (in all 10 counties) since anthracite mining was present in only two. A similar but not as dramatic a change occurred with "men's shirts and nightwear." Removal of two counties containing coal mining dropped female rates for three heart disease categories from values of more than ±10 sigma to less than ±2. Nevertheless, "men's shirts and nightwear" was retained as a candidate association for further testing, since its significance was still appreciable. This also added some weight to our association for bituminous coal mining.

It was decided to split one industry (copper ore mining) into two components because the smelting operation is so frequently associated with mining, introducing an obvious complication. Counties containing mining operations without smelters were taken as one set, and a new group for copper smelting was established. After the composites had been narrowed in this manner, unaggregated mortality rates were calculated. Only three of eight disease associations held up for mining, whereas six met the test for smelting.

The final test of the associations was to determine if the mortality rate for the sample counties was significantly higher than that calculated for a control set. The method consisted of matching each county in the sample set as closely as possible with a county in which the industry was not present. The matching criteria included: same state (if possible); median family income; percent foreign stock (matching country of origin if the number was greater than a few percent); percent living in urban areas; highest level of school completed; median age; population per square mile; and net migration. The control set thus contained the same number of counties as the sample group, located in the same or adjacent states and having approximately the same socioeconomic characteristics. The comparison between mean aggregated rates for the samples and their controls led to a final set of eight industries having a total of 21 diseases associated with them. For all of these, the sample was at least two standard deviations above the control for one sex and, in most cases, for both sexes.

Associations derived from nonregression sources were submitted to similar screening procedures. Very few of them held up under this method, which was not altogether surprising since the data used for the testing (i.e., county mortality rates) was the same data that had been used to generate the regression lists. It would be expected that these associations would have shown up in the regression analysis, if the mortality rates for counties where the industry was located were higher than average. It was possible, however, that the method of comparison with matched controls could show up a relative difference without absolute values being abnormally high.

It was necessary to use somewhat modified procedures because of the nature of some of our industry definitions. Viscose rayon production, for example, is only one component of SIC code 2823. Plants were located using industrial contacts. A county not containing a plant but adjacent to the county where the plant was located was also included when it was observed that mortality rates there were among the highest found for that disease and after learning that the plant was near the county border. Thus it is an advantage to know plant sites at this stage rather than knowing only that the county contains the industry somewhere.

For uranium mining (SIC 1094), mortality rates for diseases 05 through 56 were computed for eight counties. Those diseases having rates greater than two sigma above the national average were tested in matched control counties.

After the initial examination of sawmills (SIC 2421), that category was eliminated because too many counties contained it; also, it was believed that the original association came about because of the abundance of sawmills rather than because of the existence of a true relationship, since the diseases associated with the industry were quite varied.

We also attempted to determine potential associations by examining the 10 counties with the highest mortality rates for arteriosclerosis and congenital anomalies. Arteriosclerosis was studied by taking the 10 counties having populations above 10,000 that had the highest total white mortality rate for the disease. Industrial activity in these counties was examined to determine if any SIC code appeared consistently, but none was found. The same is true for congenital anomalies.

Two of the associations derived from nonregression sources survived the screening and validation procedures, making a total of 10 industries with their associated diseases appropriate for further study. The final group of screened and validated associations is listed in Table II-1 in Chapter II of this report. The selection of four industries was thought to be the maximum number for adequate study under existing time and budget constraints.

D. *HYPOTHESIS DEVELOPMENT AND TEST*

The basic case study approach for each industry-disease association involved two major steps: causal hypothesis development and causal hypothesis testing. The hypothesis development consisted of identifying a feasible industry-to-disease linkage comprised of a suspect industrial pollutant or etiological agent, a plausible pathway from the industry to the population, increased disease-specific mortality, and a credible disease etiology. The hypothesis testing involved a series of epidemiological analyses at specific sites to provide evidence that the hypothesis is valid—or invalid, as the case may be. For the most part, these analyses consisted of retrospective examinations of mortality and air and water quality patterns in relation to the source of the hypothesized toxic agent (i.e., the industrial facility), giving consideration to temporal as well as spatial associations.

As originally planned, the hypothesis development was to evolve from a detailed examination of the industry, the environment, and the disease etiology for specific sites and for the specific diseases under consideration. The hope was that this would lead to identification of an etiologic agent emitted from the industry, a feasible pathway to an exposed population, and a credible disease etiology. Early on it was found that this type of examination would, in general, lead to several hypotheses—any one of which could be valid. In order to determine which of the hypotheses was indicated by the particular site situation and to verify the possibility of an industry-related community disease problem, it was decided to include the analysis of mortality data in the hypothesis development process. By examining mortality patterns, this analysis indicated whether or not there was an association between disease and industry for the particular site under study and pointed to the hypothesis which best explained the association.

In general, both the hypothesis development and test approaches involved the same key elements, the primary difference being in the choice of sites for study. This will be discussed in greater detail in the following section; but, essentially, hypothesis development sites were located in counties with high mortality rates, while test sites were selected with no consideration given to mortality rates.

Detailed industrial characterizations identified potentially hazardous emissions or effluents. Pathways were determined from knowledge of the pollutants involved and from meteorologic, hydrologic, demographic, topographic, and aerial photography data. Etiologic relationships were determined from occupational diseases, literature searches, and suggestions by expert consultants.

The synthesis of information about the elements of the hypothesis resulted in the formulation of several alternative hypotheses as they related to specific populations at risk. Each of these hypotheses was then evaluated by examination of mortality, morbidity, and air and water quality data.

Confirmation of the etiological hypothesis resulted from the exposed area having increased mortality rates for the disease being studied, while rates from other diseases are reasonably equivalent to those in the unexposed area. Thus, examination of mortality rates from each of several disease categories demonstrates, if the hypothesis is tenable, that only the disease that has been etiologically implicated causes increased mortality in the exposed area. If several unrelated diseases are found to cause increased mortality, the etiologic hypothesis would not be reinforced due to the lack of specificity in elevated mortality rates.

Air and water quality data reflect the validity of the proposed pathway. It is evident that these data provide a confirmation of the existence of a plausible pathway. Together, health and environmental quality data determine which hypotheses are consistent with the actual data available at a particular site.

The supporting analyses to the hypothesis formulation and validation process discussed above are: (1) the industry analysis to identify emissions and effluents; (2) etiology analysis to identify possible etiologic agents; (3) pathway analysis to identify possible pathways and exposed populations; (4) air and water quality analysis to confirm probability of population exposure to toxicants; and (5) mortality and morbidity analysis to determine disease prevalence patterns. These five analyses each contributed to the hypothesis synthesis and validation; the magnitude of their contribution was considered as criteria for acceptance or rejection of a hypothesis.

It should be noted that each investigation differed slightly in the hypothesis development and testing procedures, although they all followed the same general procedure outlined above. These differences resulted from variations in the type, quality, and availability of data at each study site.

E. SITE SELECTION

The major assumption governing the selection of sites was that the inclusion of large metropolitan areas would substantially confuse, confound, and bias the study's results. It was felt that the possible effects of urban living, nonindustrial pollution, and industrial clutter would seriously compromise the study's efficacy. Furthermore, given the existence of suitable demographic data for nonmetropolitan areas, it really was not necessary to include large cities in the study. The ultimate effect of this decision has been discussed in Chapter III of this report.

For hypothesis development, study sites were selected on the basis of both high mortality rates and high industrial activity. After a hypothesis, consisting of an industry-originated etiologic agent and a plausible pathway, was developed, then study sites were selected for hypothesis testing on the basis of industrial characteristics only. Thus, the complete process required two successive site selections, with the second being performed only after a plausible hypothesis was determined at the first site.

The general criteria used in the selection of the hypothesis development sites were:

- High industrial activity for the study industry.
- Existence of sufficiently large population at risk.
- Lack of industrial clutter in or near the site.
- For the disease of interest, age-adjusted, sex-specific county mortality rates statistically significantly higher than the national average.

The criteria used in the selection of the hypothesis test sites were:

- For the study industry, presence of high industrial activity and emission characteristics that coincide with the hypothesis being tested.
- Existence of sufficiently large population at risk.
- Lack of industrial clutter in or near the site.

In addition, to provide a reasonable comparison for the study areas designated as "exposed," similar nearby areas, presumably unexposed, were chosen as controls. Control sites were selected to match the study sites in terms of population size, demographic characteristics, and location.

The analytical procedure for selecting the final matched control sites from the group of preliminary sites used detailed census data—i.e., age, family income, residence in 1965, race, education, sex, nativity, heating fuel, and number of persons per room in housing units—to match study and control areas on the basis of socioeconomic characteristics. The procedure utilized a weighted average of least squares deviations to compare the census statistics for study and control sites. The site which was the closest match, over all weighing schemes, was selected.

F. DATA COLLECTION

A major effort in this project has been to identify and acquire the necessary secondary data. The types of data acquired were mortality and morbidity, air and water quality, industrial processes, and demographic.

In general, mortality data were obtained from State Vital Statistics Offices, either as hard copy or on computer tape. While computerized data were obtained in less time and required less effort to prepare for analysis, they also provided less information than did copies of death certificates.

Air and water quality data were obtained from State Air Quality Reports, EPA's AEROS Data Systems, State Departments of Health, EPA's STORET System, local engineers and sanitarians, regional EPA offices, and individual industrial facilities.

The best source of industrial data was the particular company itself. About half of the companies we contacted provided process descriptions. The EPA and U.S. Department of Interior libraries supplemented this information.

Demographic data were readily available for all study areas from the Census Bureau. We obtained Fifth Count tapes from the 1970 Census for all the areas under study, and we also obtained the enumeration district maps from the Census Bureau which enabled us to correlate the street address or town of residence to a specific population at risk.

G. *INDUSTRY CHARACTERIZATION*

A principal effort in each of the case studies was to characterize the specific industries under investigation in terms of processes and pollution sources, with particular emphasis on historical trends. Information was sought from various sources including trade literature, trade associations, federal, state and local agencies, and the plants themselves. Of particuar value were EPA literature and data bases, e.g., NEDS, and EPA reprts concerned with developing point-source guidelines for specific industries. In acquiring information from specific industrial plants some plants were very helpful while others simply ignored our requsts. State and local health departments were cooperative in supplying site-specific information, but generally the information was limited. In some cases the local Chambers of Commerce were particularly useful in providing information on plant history.

H. *PATHWAY ANALYSIS*

In the pathway analysis a mechanism was postulated for each potential association of industrial pollutant and health effect, by which the pollutant might have been transported from the source to the affected population. Airborne or waterborne transport was considered, according to the type of emission.

For airborne transport, some evidence was available from air quality monitoring in the presumed area of population exposure. Generally, however, it was necessary to estimate the pathway and the intensity of exposure from historic meteorologic data. The most useful data were from the STAR (STability ARray) analyses prepared by the National Weather Center in Asheville, North Carolina. These data present wind speed and direction for various classes of atmospheric stability and can therefore be used, with a diffusion model, to estimate ground level concentration patterns in conditions favoring air pollution. STAR data are available off-the-shelf for some observing stations and are prepared to order for others. Unfortunately they are available only for major reporting stations (normally commercial airports), and the nearest station to a study site might be 50 to 100 miles distant and in different terrain. In addition, source strengths have usually to be estimated from very scanty data, especially for periods well in the past. In such circumstances it is not permissible to attach any more weight to calculated exposures than an indication whether significant airborne exposure can be plausibly hypothesized or not. Appendix A describes the atmospheric dispersion methodology used in the case studies to provide a basis for estimating the plausibility of exposure.

For waterborne transport, there are two types of pathways that can sometimes be very clearly defined: in surface streams and in underground aquifers. These can furnish unequivocal evidence for or against a potential pathway: for example, industrial effluent into a river can be assumed not to affect an upstream potable water intake but likely to affect one downstream. If underground water is withdrawn by individual private wells the picture may be much less clear, with source and pathway in doubt and usually no useful evidence from sampling. Where appropriate we obtained information on stream and other water sampling, and on geologic characteristics, from the U.S. Geological Survey or from State organizations.

I. *ETIOLOGY ANALYSIS*

The etiologic basis for industry-disease associations was investigated by means of literature searches and communications with medical experts. Relevant literature on the toxicology, epidemiology, and pathophysiology of diseases of interest was identified through computerized and manual searches and photocopied for reference throughout the study period. Additional information on emission characteristics of the suspect industries and occupational exposure within them was also collected.

J. AIR AND WATER QUALITY ANALYSIS

In analyzing air quality, our first approach was to establish temporal and spatial trends in ambient concentrations of pollutants for as many years back as the monitoring stations in the areas of interest had covered. A visual screen of the data indicated any extraordinary levels of pollutants. For those substances identified, graphing of the concentrations was done to set up visual aids for the purposes of hypothesis development.

In taking this approach, we were aware of two inherent potential difficulties. First, air quality data come from monitoring stations that are not necessarily located near the populations. It is difficult to establish from several stations whether they represent high, low, or average concentrations of the pollutants of interest, since meteorologic factors have not been taken into account. Second, monitoring station data may not reflect the concentration to which the population at risk, if any, is being exposed. For example, monitors located several miles from a plant may be measuring substances in the stack plume, while the population surrounding the plant may be exposed mainly to fugitive emissions that are not related to stack emissions.

The second approach taken in the air quality analysis was the establishment of a scenario describing how the atmospheric condition affects the dispersal of pollution from the stacks. This scenario was based on wind speed and direction data and atmospheric stability information which was obtained from the National Weather Service.

Ideally, one might have hoped to obtain a quantitative figure for the dosage of a particular pollutant experienced by the population at risk over a 10- to 50-year exposure period. This was not feasible for many reasons—foremost among them being the paucity of data available. Concentrations and absolute values of pollutants going up a stack were obtainable from stack samples and/or emission inventories. Evaluating their behavior in the environment is far more complex, due to variability of the atmosphere. The monitoring stations in the population centers we investigated, if present at all, had been established within the last several years and thus were not capable of providing us with data on earlier levels.

Investigation of water quality consisted primarily of analysis of chemical water quality of the potable water sources. The source of potable water (surface or underground) was identified, and then data on water quality were obtained for as far back in time as was available. We then determined the magnitude of the parameters and examined the data for temporal and spatial trends.

For the analysis of community water supplies coming from underground sources, the initial step in the analysis of the well water quality data was to visually scan the data for each well and evaluate whether there had been any temporal trend for each chemical parameter. Data were tabulated for the water quality parameters measured for each well. The wells were grouped by the following priorities: (1) town or population served, (2) proximity to one another, and (3) proximity to industrial operations. For those wells that had a systematic variation for certain parameters, graphing was done and slopes of the linear least squares line determined. Due to seasonal variations in concentration, the month(s) in which the data were collected was included on these graphs. These values were compared with linked parameters (such as TDS and sulfates) from the same wells, and values from other wells in the same well field, other sources supplying the same town, and other wells in industrial and control areas.

In order to link the wells with the industry and the population served, the following information was obtained:

- Length of time the well had been in operation.
- Draw depth of the well for the time period of interest.
- Type of underlaying strata for each well.
- Extent and type of casing.
- Importance of each well in supply distribution system.
- Service area of population served, present and historical.

F. MORBIDITY/MORTALITY ANALYSIS

Morbidity data were available at two sites. These data were collected from hospital records and cancer registries and analyzed for differences in number of disease-specific admissions between study and control areas.

The mortality data analysis considered principally the strength of the association between exposure and mortality. The strength of the association was assessed by examining the magnitude of age-specific crude mortality rates and summary statistics, such as standardized rate ratios. In addition, statistical significance testing of differences between standardized rates and the Mantel-Haenszel procedure for obtaining a summary relative risk over age categories were used. We also calculated the number of deaths that might be expected in a study area based on its age distribution and the age-specific mortality rates in the unexposed area.

For the purposes of determining disease specificity, the case studies examined the mortalities for 34 cause-of-death categories. These 34 categories include nearly all of the 56 cause-of-death categories examined in the SSI regression analysis, but they represent a different method of disease categorization. Reference is made to Appendix J for the relationship between the cause-of-death categories and the ICDA disease codes.

Chapter V

COPPER SMELTING CASE STUDY

A. *INTRODUCTION AND SUMMARY*

1. *Study Objectives*

The objective of this case study is to perform field investigations to explore the relationship between the copper smelting industry and community disease, in particular, digestive, respiratory, and cardiovascular diseases. The association between these diseases and the copper smelting industry was generated by the multiple regression analysis which correlated industry data with mortality data for U.S. counties [1]. The results of this present case study will assist in the evaluation of the effectiveness of the multiple regression analysis for identifying true associations between industry and community disease.

We wish to determine if the diseases originally associated with the industry in the regression analysis might be caused by the industry or might be due to other factors. We also wish to determine if there are other health problems in the community which were not identified by the regression analysis. Finally, we wish to evaluate methodology for use in the objective identification and exploration of community health problems caused by industrial pollution.

2. *Study Approach*

The approach consisted of selecting study sites and collecting the health and environmental data necessary to test the hypotheses. In this particular investigation, we tried to evaluate as many primary copper smelting sites as possible in order to provide replication and overcome the problems posed by the small populations of smelter towns. We also investigated diseases other than those found by the regression analysis to assist in determining the specificity of the original associations.

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[1] System Sciences, Inc., *Investigation Into the Industrial Correlates of Environment-Related Mortality*, 1976.

Thus, we collected mortality records for 34 disease groupings in order to permit identification of diseases missed by the regression approach. Only the data for white females were used, since many of the males in the smelter towns work in the industry and hence are exposed to an entirely different environment for one-fourth of the time. Mortality rates, actual numbers and expected numbers of deaths, standardized mortality ratios (SMRs), relative risks as compared to control areas, and statistical significance were examined. Mortality trends were then integrated with the pollution data in order to determine if relationships between the copper smelting industry and community disease existed.

The eight sites studied were:

Hayden, Arizona (Gila County)
  American Smelting and Refining Company
  Kennecott Copper Corporation

Ajo, Arizona (Pima County)
  Phelps-Dodge Corporation

Douglas, Arizona (Cochise County)
  Phelps-Dodge Corporation

Morenci, Arizona (Greenlee County)
  Phelps-Dodge Corporation

San Manuel, Arizona (Pinal County)
  Magma Copper Company

Miami-Inspiration, Arizona (Gila County)
  Inspiration Consolidated Copper Company

Anaconda, Montana (Deer Lodge County)
  Anaconda Company

Magna, Utah (Salt Lake County)
  Kennecott Copper Corporation

These sites represent eight of the total of 15 primary copper smelters in the United States. The objective of choosing several sites for this particular study was an attempt to achieve greater reliability through replication. The ideal situation, of course, would have been to examine data for all 15 sites, an impossibility under the scope of the present study.

The selection of the eight sites was governed more by practical than by technical considerations. In this study, our starting point is already existing data; thus, the sites most feasible to examine are those for which the necessary data are readily available. Thus, in the case of six of the sites, the central data collection was done by the Arizona Department of Health Services. Acquisition of information on six sites was in this case much simplified. Anaconda was chosen because of the ready availability to us of a large body of health and pollution data collected by state, local, and federal agencies. Magma in Utah was included because data on it were available as a result of the steel case study (see Chapter VI).

3. *Key Findings*

Four hypotheses pertaining to industry-disease relationships were formulated after the integration of mortality and pollution data. They are:

- Elevated mortality from acute respiratory diseases in Arizona smelter towns appears to be related to a combination of high sulfur dioxide and high particulates.
- High arsenic and cadmium from smelters in Anaconda may account for the excess mortality from cancer of the colon and pancreas.
- Excess mortality from cerebrovascular diseases in Anaconda can be explained by high levels of ambient cadmium.
- Digestive diseases, in particular cirrhosis of the liver, are most likely a result of excess alcohol consumption, although the synergistic effects of cadmium, lead, and arsenic cannot be ruled out.

B. BACKGROUND

1. Basis for Selection of the Copper Smelting Industry

In the original regression analysis performed by System Sciences, Inc. (SSI), copper ore mining (SIC 102R) was found to be associated with several disease categories:

- Respiratory diseases—bronchitis, emphysema, asthma, and acute respiratory diseases such as pneumonia, influenza, pleurisy, edema, and bronchiectasis (ICDA* 460-519,490-493)
- Cardiovascular diseases—myocardial infarction (ICDA 410-411)
- Liver diseases—cirrhosis of the liver and alcoholic cirrhosis (ICDA 571)
- Digestive diseases—nonmalignant diseases of the digestive system such as diseases of the esophagus, stomach, duodenum, intestine, peritoneum, gallbladder, and pancreas (ICDA 520-577)

Although the association was for copper mining, not smelting, we questioned the validity of using the copper mining industry as a starting point for a study of community pollution for several reasons:

- It has no obvious toxic emissions or effluents, other than ambient dust.
- Mining operations are frequently located some distance from population centers.
- Mining is very often associated with nearby smelting operations, which are notorious producers of pollution and hence a more logical source of community health problems.

Furthermore, in the original regression methodology, copper smelting had been grouped into a large, heterogeneous category of industries and had *not* been analyzed as a separate industry. Therefore, it was quite likely that mortalities actually associated with the smelting industry were obscured due to the aggregation, and that the mortalities observed for mining were actually related to the smelting operation. This reasoning prompted us to focus on the copper smelting industry.

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*ICDA codes from the *Eighth Revision International Classification of Diseases*.

2. *Overview of the Copper Industry*

Copper occurs free in nature as native copper, the predominant ore being the sulfide. The ore is mined by both open-pit and underground means. Since mined ore contains at most 6% copper, extensive extraction procedures are required. The processes used vary, depending on the nature of the particular ore. In general, they involve crushing, concentration by flotation, roasting at 600° to 800°C to remove part of the sulfur, and finally smelting at 1100° to 1600°C to produce matte. At this stage, the matte is about 30% copper sulfide and iron sulfide. The sulfur is removed by air treatment in a converter, and the iron is removed as oxide slag by addition of a siliceous flux. The blister copper is then further refined to remove traces of sulfur, iron, and precious metals in a reverberatory furnace or by electrolyte refining. A schematic of the processes is given in Figure V-1. A more detailed description of the industry is given in Appendix B of this report.

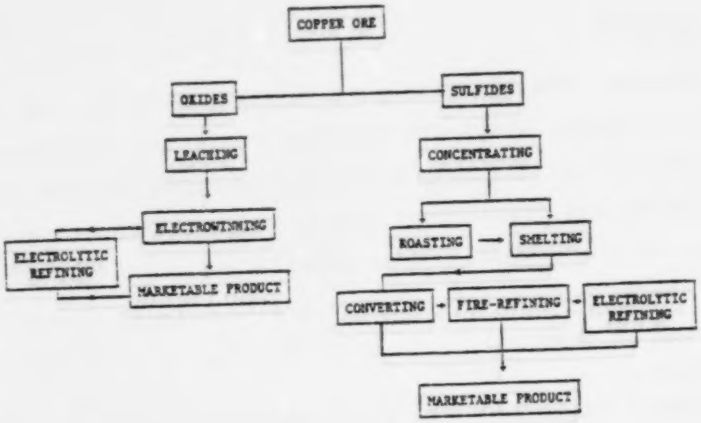

Figure V-1. Primary Copper Processes

During the process of smelting, sulfur dioxide and particulates containing heavy metals (such as lead, cadmium, and arsenic) are released. Until the 1970's, pollution was largely uncontrolled; however, recent regulations (ca. 1970) have required the industry to control the levels of emissions by the use of antipollution equipment such as electrostatic precipitators and sulfuric acid plants. The amount of uncontrolled emissions produced is huge. For example, the Kennecott smelter at Hayden produces almost 200 tons of sulfur dioxide per day and about 1 ton of particulates per day [2]. For every ton of copper produced, 0.07 pounds of cadmium and 5 pounds of arsenic are released [3]. A typical smelter can produce hundreds of tons of copper a day.

3. Health Hazards Associated with Emissions from the Industry

There are no "diseases of copper smelters," per se, although epidemiological and toxicological data suggest the plausibility of a cause-and-effect relationship between certain emissions and diseases.

Some specific adverse effects are summarized below. A detailed and documented review of the literature is given in Appendix C.

a. Sulfur Dioxide

Sulfur dioxide combines with particulates, moisture and/or oxidants to form acid sulfates, which are known to aggravate respiratory diseases such as asthma and chronic bronchitis and to cause reduced lung function and irritation of the respiratory tract. Much literature exists that reports associations between sulfur dioxide pollution and increased mortality from respiratory diseases. The health effects appear to be related to length of exposure.

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[2] EPA, Research Triangle Park, N.C., Office of Air Quality Planning & Standards, *Emission Factors for Trace Substances*, EPA-450/2-73-001, Research Triangle Park, N.C., December 1973.

[3] Effluent Guidelines Division, Office of Water and Hazardous Materials, EPA, *Development Document for Interim Final Effluent Limitations Guidelines and Proposed New Source Performance Standards for the PRIMARY COPPER SMELTING SUBCATEGORY AND THE PRIMARY COPPER REFINING SUBCATEGORY OF THE COPPER Segment of the NONFERROUS METALS MANUFACTURING Point Source Category*, EPA 440/1-75-032-b, Washington, D.C., February 1975.

The action of sulfur dioxide appears to be related to the presence of particulates. In a statement prepared for Congress, the National Academy of Sciences said that:

> "Sulfur dioxide acting alone is unlikely to be physiologically important, except during periods of severe inversion coupled with grossly inadequate emission control. However, sulfur oxide concentrations that are within the range of ambient concentrations not infrequently experienced in polluted areas of the United States may produce effects by one or more of the following mechanisms:
>
> 1. A direct effect of ambient sulfuric acid.
> 2. A direct effect of ambient suspended particulate sulfates.
> 3. An effect of sulfur dioxide potentiated by adsorption onto inert particles of respirable size.
> 4. An effect of sulfur dioxide potentiated by metallic particles that catalyze its oxidation in the airway.
> 5. An effect of sulfur dioxide potentiated by simultaneous inhalation of ozone.
>
> Inasmuch as the various sulfur oxides tend to be present together in polluted air, the effects described above may well occur simultaneously in an additive, or perhaps synergistic, manner." [4]

The synergism between particulate matter and sulfur dioxide in chronic effects on the lung is thought to be mediated via adverse effects on physiological clearance mechanisms [5].

At annual mean concentrations of 0.04 ppm, the frequency of lung disease may increase and mortality from bronchitis and lung cancer may occur. At concentrations of 0.2 to 0.3 ppm, patients with chronic lung disease may experience accentuation of symptoms, and the general population may experience increased mortality [6]. At concentrations greater than 1 ppm, sulfur dioxide causes nausea and coughing, even in persons without preexisting respiratory disease. The taste threshold is at 0.3 ppm. The relationship between length of exposure and concentration of SO_2 is shown in Figure V-2 [7]. The air quality standards for the three states studied—Arizona, Utah, Montana—are shown in Table V-1.

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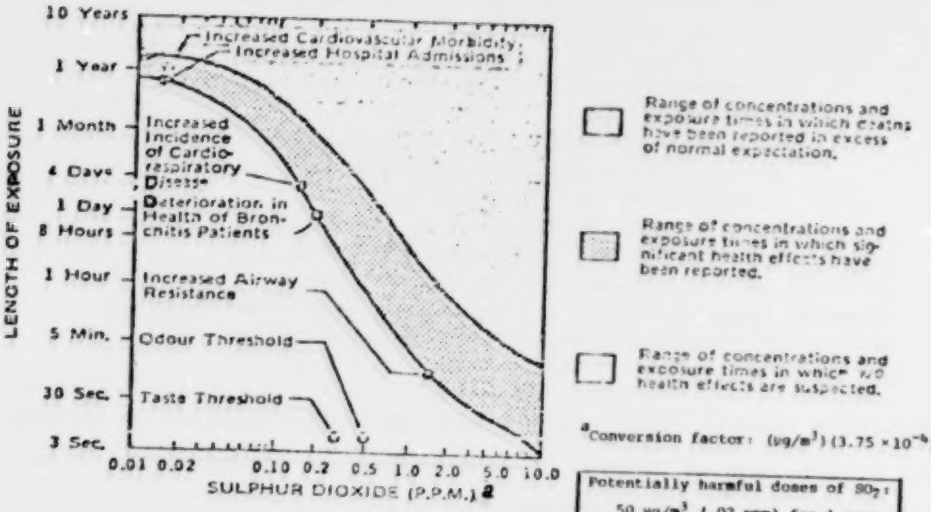
[4] *Air Quality and Stationary Source Emission Control*, A Report by the Commission on Natural Resources, National Academy of Sciences, National Academy of Engineering, National Research Council, Prepared for the Committee on Public Works, U.S. Senate, Serial No. 94-4, March 1975.

[5] *Proceedings of the Conference on Health Effects of Air Pollutants*, Assembly of Life Sciences, National Academy of Sciences-National Research Council, October 3-5, 1973, Prepared for the Committee on Public Works, U.S. Senate, Serial No. 93-15, November 1973.



[a] Conversion factor: $(\mu g/m^3)(3.75 \times 10^{-4}) = ppm$

Potentially harmful doses of SO_2:
50 $\mu g/m^3$ (.02 ppm) for 1 year
240 $\mu g/m^3$ (.09 ppm) for 1 month
800 $\mu g/m^3$ (.30 ppm) for 1 day

Figure V-2. Health Effects of Sulfur Dioxide Pollution [7]

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[6] HEW, Consumer Protection and Environmental Health Service, National Air Pollution Control Administration, *Air Quality Criteria for Sulfur Oxides*, NAPCA Publ. No. AP-50, January 1969.

[7] Barringer, Health effects of sulfur dioxide pollution, in *Correlation Spectrometry Applied to Earth Resources*, Princeton University Conference, June 1970.

Table V-1

AMBIENT AIR QUALITY STANDARDS: SULFUR DIOXIDE & PARTICULATES—ARIZONA, UTAH, MONTANA, FEDERAL

ARIZONA STANDARDS[a]

POLLUTANT	AVERAGING PERIOD	AMBIENT AIR QUALITY STANDARDS
Sulfur Dioxide	Annual	50 $\mu g/m^3$
	72-hour	120 $\mu g/m^3$
	24-hour	250 $\mu g/m^3$
	1-hour	850 $\mu g/m^3$
Particulates	Annual	70 $\mu g/m^3$

UTAH STANDARDS[b]

POLLUTANT	AVERAGING PERIOD	AMBIENT AIR QUALITY STANDARDS	
		PRIMARY	SECONDARY
Sulfur Dioxide	Annual	80 $\mu g/m^3$ (0.03 ppm)	None
	24-hour	365 $\mu g/m^3$ (0.14 ppm)	None
	3-hour	None	1300 $\mu g/m^3$ (0.5 ppm)
Particulates	Annual	75 $\mu g/m^3$	60 $\mu g/m^3$
	24-hour	260 $\mu g/m^3$	150 $\mu g/m^3$

MONTANA STANDARDS[c]

POLLUTANT	AVERAGING PERIOD	AMBIENT AIR QUALITY STANDARDS
Sulfur Dioxide	Annual	0.02 ppm
	24-hour	0.10 ppm
	1-hour	0.25 ppm
Suspended Particulates	Annual	75 $\mu g/m^3$
	24-hour	200 $\mu g/m^3$
Settled Particulates	3-month	15 T/mi^2 (residential area)

FEDERAL STANDARDS

POLLUTANT	AVERAGING PERIOD	AMBIENT AIR QUALITY STANDARDS	
		PRIMARY	SECONDARY
Sulfur Dioxide	Annual	80 $\mu g/m^3$	60 $\mu g/m^3$
	24-hour	365 $\mu g/m^3$	260 $\mu g/m^3$
	3-hour	No Standard	1300 $\mu g/m^3$

[a] Arizona Department of Health, Division of Air Pollution Control, July 1972.

[b] Utah State Division of Health.

[c] Annual Air Quality Data Summary for Montana, 1976, Department of Health and Environmental Sciences, Air Quality Bureau.

b. Arsenic

Adverse effects of arsenic include anemia, gastric disturbances, renal symptoms, ulceration, carcinogenesis (poorly differentiated epidermoid bronchogenic carcinoma), and liver and common bile duct degeneration in animals. Arsenic may exert a synergistic effect with alcohol, and may be taken into the body through air, water, and food.

Maximum permissible atmospheric concentrations have not been adopted in the United States for arsenic. Water standards, however, have been adopted, and concentrations of 2 to 4 mg per liter are reported not to interfere with the self-purification of streams. The Public Health Service Drinking Water Standards state that the concentration of arsenic in drinking water should not exceed 0.01 mg/liter, and concentrations in excess of 0.05 mg/liter are grounds for rejection of the supply [3].

The magnitude of the arsenic pollution (and other heavy metals) produced by smelters is enormous. For example, it has been determined that 1 to 4 milligrams of arsenic are deposited per square meter per month within a 1-mile radius of a zinc smelter in East Helena, Montana [8]. The potential to spread to the surrounding community has also been demonstrated by the finding of a gradient of urinary arsenic in residents living downwind from a smelter. Levels were 0.3 ppm at a distance of 0.0 to 0.4 miles, and 0.02 ppm at a distance of 2.0 to 2.4 miles. Samples of vacuum cleaner dust declined from 1300 ppm at a distance of 0.0 to 0.4 miles, to 70 ppm at a distance of 2.0 to 2.4 miles [9].

NIOSH has recommended that no worker be exposed to a concentration of arsenic in excess of 2 $\mu g/m^3$ of air as determined by a 15-minute sampling period [10]. Calculations based on estimates of 8-hour time-weighted-average arsenic exposure and actual deaths from respiratory cancer mortality in exposed workers have shown an apparent dose-response relationship between arsenic exposure and respiratory cancer mortality (see Table V-2). Based on a 40-year working life, these calculations suggest that respiratory cancer mortality was twice that expected at a dosage equivalent to exposure for 40 years at 3.0 µg As/m^3 on an 8-hour TWA basis, while the observed-expected ratio was 0.6 at the equivalent to 1.0 µg As/m^3.

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[8] EPA, Office of Air Programs, Research Triangle Park, *Helena Valley Montana Area Environmental Pollution Study*, AP-91, Research Triangle Park, N.C., 1972.

[9] Milham, S., and T. Strong, Human arsenic exposure in relation to a copper smelter, *Environ. Res.* 7:176-182, 1974.

[10] HEW, NIOSH, *Occupational Exposure to Inorganic Arsenic: New Criteria*, DHEW Publ. No. (NIOSH) 75-149, NIOSH, Washington, D.C., 1975.

Table V-2. RESPIRATORY CANCER DEATHS BY EXPOSURE TO ARSENIC CATEGORY

ARSENIC EXPOSURE		Total Deaths (n=173)	RESPIRATORY CANCER DEATHS		
Average Dosage* (mg)	Projected 8-Hour TWA (µg)		Observed (n=28)	Expected	O/E
3.74	1.0	26	1	1.77	0.6
4.84	3.0	17	2	1.01	2.0
5.53	6.0	24	4	1.38	2.9
6.04	10.0	22	3	1.36	2.2
6.68	20.0	27	3	1.70	1.8
7.35	40.0	18	2	0.97	2.1
8.17	90.0	13	3	0.77	3.9
8.78	160.0	13	5	0.79	6.3
10.30	740.0	13	5	0.72	7.0

Source: Reference [10].
*Total dose based on a 40-year working life.

c. *Cadmium*

Cadmium is a highly toxic element affecting many systems. It is apparently absorbed without regard to the bodily level of cadmium, indicating a lack of homeostatic mechanisms for the control of cadmium levels. Adverse effects include pulmonary emphysema, renal dysfunction (proteinuria), anemia, and bone and tooth disease. It may also have carcinogenic [11] and gastrointestinal (including liver) effects. Its implication in hypertensive disease has been shown through animal experiments and epidemiological evidence; that is, high levels of cadmium have been found in body tissues of those who died from hypertensive disease.

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[11] Sunderman, F. W., Carcinogenic effects of metals, *Fed. Proc.* 37(1): 40-46, 1978.

High serum levels were also found in residents of a steel city suffering from cardiovascular disease. In a study on the effects of chronic exposure of rats to cadmium, rats received, from time of weaning, drinking water containing 5 ppm of cadmium. Hypertension began to appear after about a year and the incidence, which was greater in females, increased with age. Both sexes had decreased median life spans compared to cadmium-free controls. In this study, the renal concentration of cadmium found in the rat kidney was less than that reported for human kidney. Thus, one form of hypertension in man may be related to the accumulation of cadmium in the kidney, probably in the renal cortex, where cadmium is firmly chelated to a zinc- and cadmium-containing protein [8].

Maximum permissible atmospheric concentrations for cadmium have not been adopted in the United States. In 1963, the National Air Sampling Network reported that the nation's air contained an average 24-hour concentration of 0.002 to 0.037 $\mu g/m^3$ in urban areas and from 0.0004 to 0.026 in nonurban areas. The highest value in the survey was 0.050 $\mu g/m^3$ in Covington, Kentucky. Standards have, however, been set for water. According to the Public Health Service Drinking Water Standards, a drinking water supply containing in excess of 0.01 mg of cadmium per liter should be rejected.

A summary of the effects of varying levels of cadmium exposure is given in Table V-3 [12].

d. *Lead*

Adverse effects of lead include weakness, anemia, neurological and psychiatric disturbances, and gastrointestinal dysfunctions. It may be taken in through air, water, or food routes.

The effects of chronic exposure of rats to lead were studied by giving rats 5 ppm of lead in drinking water from weaning until death. Lead exerted a continuous adverse effect at all ages and in both sexes

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[12] HEW, Center for Disease Control, NIOSH, *Criteria for a Recommended Standard. Occupational Exposure to Cadmium*, HEW Publ. No. (NIOSH) 76-192, Washington, D.C., August 1976.

Table V-3. SUMMARY EFFECTS OF CADMIUM EXPOSURE

SPECIES	ROUTE	DOSE or CONCENTRATION	EXPOSURE	COMMENTS
Occupational Exposure in Humans*				
Humans	Inhalation	2500-2900 mg-min/m^3	Once	Fatal pulmonary edema
Humans	Inhalation	0.1-25 mg/m^3	Years	Rickets and dental problems in offspring of workers
Humans	Inhalation	0.1-24 mg/m^3	Years	General complaints, chronaxy changes
Humans	Inhalation	0.04-19 mg/m^3	Years	Slight anemia, yellow fringe on teeth
Humans	Inhalation	3-15 mg/m^3	Years	Anosmia, fatigue, renal dysfunction, hepatic dysfunction, emphysema, yellow fringe on teeth
Humans	Inhalation	0.14 mg/m^3	Years	Emphysema, proteinuria
Humans	Inhalation	0.6-2.8 mg/m^3	Years	Anosmia, proteinuria
Humans	Inhalation	0.028-2.8 mg/m^3	Years	Anosmia, proteinuria
Humans	Inhalation	0.33-1.9 mg/m^3	Years	Proteinuria, aminoaciduria
Humans	Inhalation	20-700 μg/m^3	Years,1 hr/day	Proteinuria
Humans	Inhalation	60-680 μg/m^3	Years	Gastrointestinal and respiratory symptoms, carious teeth
Humans	Inhalation	80-450 μg/m^3	Years	Emphysema, proteinuria
Humans	Inhalation	74-210 μg/m^3	Years	Reduced pulmonary function, proteinuria
Humans	Inhalation	134 μg/m^3	1-20 years	Proteinuria
Humans	Inhalation	125 μg/m^3	Years	Anemia, proteinuria
Humans	Inhalation	Below 100 μg/m^3	Years	Questionable effects
Humans	Inhalation	Below 90 μg/m^3	Years	Reduced pulmonary function, proteinuria
Humans	Inhalation	66 μg/m^3	21-40 years	Reduced pulmonary function, proteinuria
Humans	Inhalation	31 μg/m^3	1-12 years	No effects
Humans	Inhalation	16-29 μg/m^3	Years	Improvement in conditions of workers exposed at 125 μg/m^3, no effects in new workers
Exposure in Animal Studies				
Rats	po, water	50 ppm	3 months	Anemia
Rats	po, diet	45-135 ppm	6 months	Anemia, bleaching of incisor teeth
Rabbits	sc	650 μg/kg	10 weeks	Anemia, proteinuria
Rats, mice	po, water	5 ppm		Hypertension after 1 year
Rats	po, water	0.2-200 ppm	6-12 weeks	Vascular changes in kidneys
Rats	ip	1 mg/kg	45 days	Increased adrenal activity
Rats	sc	850 μg/kg	Once	Testicular necrosis
Rats	po, water	5 ppm	Up to 4 years	No significant increase in tumors
Mice	sc	630 μg/kg	Once	Fetal abnormalities
Hamsters	iv	880 μg/kg	Once	Fetal abnormalities
Rats	ip	1.8 mg/kg	Once	Fetal abnormalities
Rats	Inhalation	2.8 mg/m^3	7 months	Altered estrus, decreased weights of offspring

*Some investigators noted deaths of cadmium workers from various causes, including cancer, without necessarily attributing them to exposure.

Source: Reference [12].

as evidenced by reduced life spans and increased mortality rates. Concentrations of lead in the rat organs were similar to those reported for man [8].

Lead can be seriously injurious to health as a result of accumulations in the body. Long-term daily intake of less than 0.6 mg by healthy adults may cause small increases in body burden, but no clinical disease. An intake in excess of 0.6 mg per day may result in the accumulation during a lifetime of a dangerous quantity of lead in the body.

It has been shown that the lead content of dustfall, and consequently of soil, vegetation, and outdoor dust, decreases exponentially with distance from smelters and that children living in contaminated areas absorb excessive amounts of lead [13]. The National Air Sampling Network reported a range of 0.1 to 2.3 $\mu g/m^3$ of lead in urban areas, and from 0.002 to 0.15 $\mu g/m^3$ in nonurban areas. The highest city in the 1964 NASN survey was Minneapolis with 2.7 $\mu g/m^3$. Maximum permissible atmospheric concentrations for lead have not yet been established in the United States.

The lead concentration in drinking water supplies ranged from traces to 0.04 mg per liter, averaging 0.01 mg per liter. At concentrations of 0.1 mg per liter, bacterial decomposition of organic matter is inhibited and some fish are susceptible to lead poisoning. Adults consume 1 to 3 liters of drinking water per day. The Public Health Service Drinking Water Standards state that 0.05 mg of lead per liter constitutes grounds for rejection of the water supply.

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[13] Parkinson, D. K., Lead contamination around secondary smelters: Estimation of dispersal and accumulation by humans, *Science 186*: 1120-1123, 1974.

e. *Fluoride**

The role of fluoride in the production of cancer is controversial [14]. A small amount of epidemiological and toxicological research points to its possible involvement. Experiments with mice have shown that fluoride stimulates cancer growth at concentrations as low as 1 ppm, whether taken in water or by subdermal injection or even in *in vitro* suspensions. Also, when fluoride is incorporated into the carcinogen dimethylaminobenzene, its cancer-producing ability is greatly enhanced. High cancer rates in workers exposed to fluoride have been reported, although the results are not clear-cut because radiation and carcinogen exposure were also present. In a steel city in Ontario, correlations were found between ambient fluoride levels, increased cancer rates, and skeletal fluorosis [15]. Pollution data indicate that standard maximum levels (5 ppb) for fluoride were exceeded in the early 1970's in the Canadian steel city. Levels in home grown food were 17 to 130 times the maximum permissible level. Atmospheric levels of fluoride of up to 1180 mg F/100 cm^2/30 days were observed in the Ontario steel city in 1972, a value 30 times higher than for the rest of Ontario.

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* Although not an emission from copper smelters, fluoride is discussed here because one of the areas studied has a problem with fluoride contamination as a result of another pollution source.

[14] Editorial: Can fluoride cause cancer?, *Fluoride* 5(4):169-170, October 1972.

[15] Cecilioni, V. A., Further observations on cancer in a steel city, *Fluoride* 7(3):153-165, July 1974.

4. *Description of the Study Sites*

Eight sites involving nine copper smelters were studied:

- Ajo, Arizona (Pima County)
  —Phelps-Dodge Corporation
- Morenci, Arizona (Greenlee County)
  —Phelps-Dodge Corporation
- Douglas, Arizona (Cochise County)
  —Phelps-Dodge Corporation
- Hayden, Arizona (Gila County)
  —American Smelting and Refining Company
  --Kennecott Copper Corporation
- San Manuel, Arizona (Pinal County)
  —Magma Copper Company
- Miami-Inspiration, Arizona (Gila County)
  —Inspiration Consolidated Copper Company
- Anaconda, Montana (Deer Lodge County)
  —Anaconda Company
- Magna, Utah (Salt Lake County)
  —Kennecott Copper Corporation

Maps of these areas are given in Appendix D.

Ajo, established in 1864, is located in southern Arizona, west of Tuscon. A large percentage of the labor force is employed in the mining and smelting operations of the New Cornelia Mine owned by the Phelps-Dodge Corporation directly east-southeast of town. Other occupations are largely in service and commercial trade. From 1960 to 1970, the population decreased from 7,049 to 5,881, or -16%.

Clifton-Morenci, on the eastern border of the state, was established in the 1880's and today is dominated by the copper smelter owned by Phelps-Dodge. Trade and service occupations are also represented as well as governmental activities in Clifton, the county seat. From 1960 to 1970, the population increased from 7,248 to 8,140, a 12% change.

Douglas is the largest city in Cochise County and is situated on the U.S.-Mexico border. The principal occupations are: mining and smelting, apparel manufacture, government employment, cattle transport, and agriculture. The smelter is located about 2.5 miles west of town.

Hayden is located one mile north of the junction of the Gila and San Pedro Rivers at the southern end of the county. Over 1,400 workers are employed in the town's two copper smelters. A small amount of retail trade exists and agriculture is not prominant. The population has declined 27% from 1960 to 1970, going from 1,760 to 1,283.

San Manuel, a recently established town, started in the 1950's. The population has been stable, changing only 2% from 1960 to 1970 (1,913 to 1,953). The mining and smelting facilities of the Magma Copper Company employ 3,000 people. The area also supports agriculture, cattle ranches, and guest farms.

*Miami,** established in the 1900's, is located in the Pinal Mountains of east central Arizona, between the walls of a steep canyon. Inspiration Consolidated Copper Company, Miami Copper Company, and Ranchers Exploration and Development operate open-pit and underground copper mines and leaching operations. Inspiration also operates a smelter, refinery, and rod mill. The copper industry in the Miami area employs 3,500 workers. Other industries are a lime quarry, cattle ranching, and a sawmill. The area was for many years a producer of high-grade asbestos which was mined in the area from 1913 on. In the early days most material was hand-processed, but from the mid-1940's to the mid-1970's several mills were operated in the town of Globe, about 7 miles southeast of Miami. All plants except one closed by 1974 as a result of federal safety regulations.

A detailed history of the demography and industrial activity in Gila and Pinal Counties is given in Appendix E.

Anaconda is in the Deer Lodge Valley of Montana. The valley is 35 miles long and 8 to 10 miles wide, sloping in a northerly direction from Anaconda at an elevation of a little over 5,000 feet to Garrison at an elevation of approximately 4,500 feet. The smelter at Anaconda employs about 1,700 men and processes copper and zinc ore mined in Butte, approximately 27 miles east. The majority of the population is engaged either directly or indirectly with the copper industry. The smelter is two miles east-southeast from the center of town.

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* A smelter was in operation from 1924 to 1971 in Superior, 10 miles northwest of Miami.

Magna, Utah, is located on the floor of the Salt Lake Valley, 4,220 feet above sea level and about five miles from the smelter. It is between the Wasatch Mountains to the east and the Oquirrh Mountains to the west.

The major processes and emissions from the copper smelting industry were outlined in Section B-2, and a detailed description of the industry is given in Appendix B. In Table V-4 we list the features specific to the sites under study [16].

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[16] EPA, Office of Air and Waste Management, Office of Air Quality Planning and Standards, *Background Information for New Source Performance Standards: Primary Copper, Zinc, and Lead Smelters, Volume I: Proposed Standards*, EPA-450/2-74-002a, Research Triangle Park, N.C., October 1974.

Table V-4. DESCRIPTIONS OF SELECTED COPPER SMELTERS*

COMPANY	AGE OF PLANT		MATERIALS		PRODUCTION EQUIPMENT						STACK DATA					
					ROASTERS		SMELTING FURNACES		CONVERTERS						EMISSION RATE	
NAME/ LOCATION	FIRST YEAR	LAST MODIFICATION	CONC. (t/d)	BLISTER COPPER (t/d)	# of OPER/ STBY	GAS STREAM CONTROL EQUIPMENT	# of OPER/ STBY	GAS STREAM CONTROL EQUIPMENT	# of OPER/ STBY	GAS STREAM CONTROL EQUIPMENT	NUMBER	HGT. (ft)	FLOW RATE (acfm)	SO_2 CONC. (ppm)	PARTIC. (t/d)	SO_2 (t/d)
ASARCO/ Hayden, Arizona	1912	1971 (New converter acid plant)	2,000	366	7/5	Roaster gases join reverb gases prior to treatment.	2/0	Reverb gas thru WHBs and water spray chamber, then joined with roaster gases to ESP (98.3%) and 300-ft stack.	4/1	All converter gases to 1970 Rust Engineering 750 t/d acid plant.	1 (roast., furnace) 1 (tail gas)	300 30	401,000 81,200	5,900 2,200	1.5 0	290 21.8
Magma/ San Manuel, Arizona	1956	--	1,700	310 (Anode Cu)	None	--	2/0	Reverb gases treated in WHB and ESP (89%), then out 515-ft stack.	5/0	Converter gases passed thru ESP (95%), then to 550-ft stack.	1 (furn.) 1 (conv.)	515 550	175,000 117,000	15,000 54,000	1.79 0.54	313 767
Phelps-Dodge/ Douglas, Arizona	1910	1971 (New ESP	2,260	365 (Anode Cu)	17/7	Roaster gases treated by ESP and then join reverb gases.	3/0	All reverb gases treated in WHB, then join roaster gases and pass out 544-ft stack.	3/2	Converter gases treated in ESP, then passed out 563-ft stack.	1 (roast., furn.) 1 (conv.)	544 556	470,000 264,000	15,700 15,200	39 9	902 492
Phelps-Dodge/ Morenci, Arizona	1942	1964 (Roaster acid plant)	2,113	470 (Anode Cu)	1/0	All roaster gases to Parsons 750 t/d acid plant.	4/0	Reverb gas treated in two ESPs in parallel (78.5%).	8/0	Converter gases treated in ESP (98.5%).	1 (tail gas, furn.) 1 (conv.)	600 600	310,000 406,000	14,000 15,400	14.7 0.8	526 768
Phelps-Dodge/ Ajo, Arizona	1950	1972 (DMA & acid plants)	680	197 (Anode Cu)	None	--	1/0	Reverb gases join converter gases, then to ESP and 360-ft stack.	2/0	Converter gases join reverb gases, then to ESP and 360-ft stack.	1 (furn., conv.)	360	397,000	7,000	0.7	380
Inspiration/ Miami, Arizona	1915	1972 (Elect. furn., syph. conv., acid plt. under const.)	840	300	None	--	1/0	Reverb gases pass thru WHBs, flue, and 275-ft stack	3/1	Converter gases treated by balloon flue to 200-ft stack	1 (furn.) 1 (conv.)	275 200	260,800 163,400	2,500 15,200	Unk. Unk.	78 296
Kennecott/ Hayden, Arizona	1958	1968 (Fluid bed roaster and acid plant)	1,050	220	1/0	All roaster gases pass thru cyclones & ESP (50%), then join 50% of converter gases & pass thru 1968 Monsanto 750 t/d acid plant.	1/0	Reverb gases treated by WHB, mixed with 50% of converter gases, passed thru ESP (95%) and 600-ft stack.	2/1	Converter gases pass thru balloon flue, then: 50% of gases mixed with reverb gases, and 50% of gases treated in ESP (95%) and added to roaster gases	1 (tail gas) 1 (furn., 50% conv.)	100 600	75,000 211,000	1,300 7,250	0 1.1	12 187
Kennecott/ Magna, Utah	1907	1968 (Removed roasters and converted to green-feed reverbs)	2,200	750	None	--	3/0	Reverb gases treated by WHB and ESP (50%), then out two 410-ft stacks.	7/2	All converter gases enter ESP (60-70%), then 97.5% of gases treated in five acid plants (1,400 t/d H_2SO_4), and out five acid plant stacks.	2 (furn.) 5 (tail gas)	408 95, 95, 87, 82, 126	1,107,000 259,000	3,500 1,000	4.33 0.46 (acid plant by-pass flue)	323 32
Anaconda/ Anaconda, Montana	1906	1973 (New acid plant)	1,710	500 (Anode Cu)	None	Roasters exist, but for drying concentrate only.	3/1	Reverb gases pass thru water spray chamber and flue, then join converter gases.	6/0	Converter gases join reverb gases, pass thru settling flue and ESP (36.9%), then out stack.	1 (furn., conv.)	925	1,950,000	3,300	22.5	790

*Source: Reference (16)

C. APPROACH

1. General Methodology

a. Analysis of Mortality Data

Data interpretation was based on the results of the mortality analyses. We began with the objective of determining whether or not there was an excess of mortalities in the study areas. This involved establishing criteria to screen rates and designate them as elevated or not elevated. Because of the small populations involved in many of the towns of interest, we did not look at mortality rates alone. Since mortality rates are standardized to a population base of 100,000, it is possible for a particular rate to be misleadingly high when, in fact, only a very few deaths (e.g., two or three) are involved. We therefore identified disease categories having elevated rates by a combination of several measures. We considered a rate to be elevated if it met the following criteria:

- higher than the national average and one or more comparison areas;
- standardized mortality ratio (SMR) of over 100; and
- $p < 0.05$ as determined by the Mantel-Haenszel test of significance [17]; that is, relative risk >1 and $\chi^2 > 3.85$.

The rates analyzed were those of white females.* We based our conclusions on female rates because, in the towns studied, a large proportion of the males are workers at the smelter. Smelter workers are exposed, for a fourth of the time, to a situation where the pollution conditions are very different from those outside the plant. Males, therefore, represent a mixed-exposure situation and were not considered in this approach.

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[17] Mantel, N., and W. Haenszel, Statistical aspects of the analysis of data from retrospective studies of disease, *J. Natl. Cancer Inst.* *22*(4):719-748, 1959.

* The demographic category "white" may include those of Hispanic stock. To separate those of Hispanic origin would require evaluation of country of origin and/or surname data from death certificates; this information is not coded on the mortality tapes provided by the states.

Furthermore, even in towns where the majority of males are employed in the smelting operation, they do not represent as homogeneous a study population as do females. It is frequently the case that men employed in the copper industry have had previous employment histories in other types of industry with potential exposure to hazardous substances. This history would represent a potential confounding factor in the study and could not be known without conducting a case study of individual residents.

Although the original regression analysis identified a group of seven diseases, we obtained and analyzed the rates for some 34 disease groupings. We felt that an analysis of all rates would enable us to verify the results of the original regression and help in an evaluation of its usefulness. This did not entail any extra cost or effort in the acquisition of data, since the rates for all diseases for a particular area were included on the same tape. It did, of course, entail more effort in the actual analysis and etiology research.

The original plan submitted for this program called for the investigation of only two sites—one for hypothesis development and the other for hypothesis testing. In the copper smelting study, we wanted to examine the rates for as many sites as possible to permit replication, and as a compensation for the small populations frequently found in company towns. It was ambitious to evaluate eight sites in a time frame originally designed for two sites. Consequently, it was necessary to sacrifice depth for breadth in certain aspects of the study. We felt at this stage that it was more important to determine if a number of copper smelter towns had elevated mortalities for the same disease rather than to scrutinize one town in great depth. After a beginning of this type, we envisaged that the next step in the long-range plan would be to study each town in detail.

Because of the small populations and few deaths, it was necessary in some parts of the analysis to aggregate disease groups and towns in order to have an adequate number of data points. Therefore, in Arizona the only town large enough to analyze separately was Douglas. First, the rates for individual diseases for Douglas were analyzed separately.

Second, the number of categories was decreased in order to produce more data points per category. Thus, the original 34 disease groups were collapsed into 6 and the deaths from all 6 towns were combined in one group. The aggregated data were screened by comparing deaths per population for the smelter town aggregate to the same ratio for a control area. This method is crude and not adjusted for age but is believed to be suitable to indicate broad trends.

b. Comparison Areas

The original intention was to select as many control sites as feasible, so that conclusions would not be based on comparison to only one other figure. Comparison areas were chosen which did not have smelting or another industry that is a major source of pollution, and which were reasonably similar to the study areas in terms of socioeconomic variables.

For Anaconda, five areas were identified: Billings, Bozeman, Havre, Livingstone, and Great Falls. For Magna, three were chosen: Logan, Cedar City, and Price. Conversations with local health and pollution officials in Montana and Utah confirmed the suitability of these areas.

In Arizona, the selection of control areas was more of a problem. Much of the area is dominated by nonwhite populations from Indian reservations. Furthermore, towns with significant white populations often were found to differ from study areas in socioeconomic characteristics, such as education, median age, and income. Other areas were primarily retirement areas or college towns. There are wide differences in climate throughout the state, which may have an effect on certain health problems (such as respiratory diseases) or air quality (such as the amount of particulates). Other issues were the presence of other polluting industries such as coal-fired power plants. It was finally decided to use the State of Arizona as a comparison area.* In this way, it was felt that local differences would average out.

The summary demographic statistics for the study sites and comparison areas are given in Table V-5.

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* Actual data used were for the State of Arizona minus Gila and Pinal Counties.

Table V-5

SUMMARY DEMOGRAPHIC STATISTICS: ARIZONA SMELTER TOWNS AND CONTROL AREA; ANACONDA AND CONTROLS; MAGNA AND CONTROLS

STATE	DEMOGRAPHIC AREA	DEMOGRAPHIC VARIABLES							
		Median Age	Median Family Income	% in Same House as in 1965	% White	% With Less Than 1 Person per Room	% Completing High School	% Female	% Native-Born, Native Parents
ARIZONA	Ajo (S)	29.5	9,500	41.8	89.9	83.4	49.5	51.0	72.3
	Douglas (S)	23.0	7,500	57.5	98.4	83.5	43.3	52.7	43.3
	Morenci (S)	29.5	9,500	51.1	97.2	87.5	52.0	51.2	83.6
	Hayden (S)	21.0	9,500	70.3	100.0	71.1	41.9	50.6	68.6
	San Manuel (S)	18.0	11,000	32.2	97.9	77.3	63.2	48.7	85.9
	Miami (S)	23.0	7,500	63.1	99.1	83.8	41.8	51.1	71.3
	Aggregated smelter towns	23.0	8,500	52.2	96.7	82.4	47.1	51.4	61.1
	Arizona[1] (C)	29.5	9,500	41.0	91.4	88.1	58.9	50.9	83.2
MONTANA	Anaconda (S)	29.5	8,500	59.4	98.5	92.1	53.2	51.8	72.5
	Billings (C)	29.5	9,500	48.3	98.0	94.4	68.2	52.0	82.6
	Bozeman (C)	21.0	8,500	29.5	99.2	95.2	77.2	49.2	87.1
	Great Falls (C)	29.5	9,500	48.8	97.2	92.7	66.1	51.6	81.2
	Havre (C)	23.0	9,500	48.2	96.8	91.6	66.1	50.1	80.6
	Livingston (C)	39.5	7,500	52.5	99.6	94.7	53.2	52.1	78.3
UTAH	Magna (S)	21.0	9,500	66.0	99.4	84.3	56.4	50.4	88.9
	Cedar City (C)	21.0	8,500	46.4	98.6	90.6	78.4	50.0	92.1
	Kearns (C)	16.0	9,500	64.3	99.6	77.0	53.9	50.1	92.4
	Logan (C)	23.0	7,500	39.9	97.9	93.9	80.4	50.2	86.3
	Price (C)	29.5	8,500	53.7	98.7	92.4	57.5	51.0	79.1

(S) denotes towns with smelter.

(C) denotes control town or area

[1] Data used was for Arizona minus Gila and Pinal Counties.

2. *Data Integration*

After identifying elevated mortalities by the method described above, the next step was to determine if the effect could be explained by pollution patterns. The question was: Does the excess mortality occur in areas which have pollution of a type implicated in the etiology of the disease?

The nature of the pollution data available had an obvious and important effect upon the type of analysis that could be undertaken. Several problems with the available pollution data were recognized. There is a lack of information on exactly how ambient environmental measurements relate to exposure of individuals. This is a result of (1) unanswered questions on the absorption, metabolism, and excretion of low doses of pollutants over a long period and (2) lack of detailed knowledge on the fate, transformation, interaction, and dispersal of pollutants in the atmosphere. Further complicating the issue is the influence of personal lifestyle,such as how long the individual has been exposed, his general health, diet, smoking habits, and level of health care. These gaps in the present knowledge are reflected in the fact that, for many of the pollutants released by the smelting industry, no atmospheric standards exist.

Another major drawback is the general lack of pollution data on control areas. In a rigorous analysis, one would want to compare mortality and pollution patterns in a study area to the same data in a control area. Unfortunately, much of the detailed pollution data which is presently available is from large urban areas or unpopulated expanses, both of which are unsuitable as controls.

3. *Data Collection and Data Bases*

For each of the sites examined, it was necessary to collect health, pollution, industry, and demographic information. This was a major effort and required (1) identification of the sources of the data; (2) consultations with officials to determine the cost, availability, and quality of the information; and (3) acquisition of the data, either as hard copy, magnetic tape, or by manual abstracting on site. In some cases there were gaps or redundancies among the various sources; we often found that measurements for a particular time, location, or pollutant, which would have been useful in the analysis, simply did not exist.

The following subsections summarize the major data sources compiled for the study.

a. *Mortality Data Bases*

ARIZONA 1968-1975

YEAR	COUNTIES	CAUSES OF DEATH	PLACE OF RESIDENCE OF DECEASED	LENGTH OF RESIDENCE	
				ARIZONA	TOWN
1968	All Arizona Counties	All	Yes	Yes	Yes
1969		↓	↓	↓	↓
1970					
1971	↓				No
1973					↓
1974					
1975					

Note: The core of this data base was acquired on tape from the Arizona Department of Health Services (ADHS). Note that Arizona listed length of residence in the town on death certificates for only three years, although length of residence in the state is listed for all years.

GILA & PINAL COUNTIES 1957-1975

YEAR	COUNTIES	CAUSES OF DEATH	PLACE OF RESIDENCE OF DECEASED	LENGTH OF RESIDENCE		OCCUPATION
				ARIZONA	TOWN	
1957	Gila & Pinal	All	Yes	Yes	No	Yes
1960		↓	↓	↓	↓	↓
1963	↓					
1966						
1969						
1972						
1975						

Note: These data were obtained as hard copy from the ADHS. All data contained on the certificates was coded and punched in-house and put on tape. If the case of 1969-1975, it was necessary to code and punch occupational information only since the other variables are available on the ADHS tape.

GILA & PINAL COUNTIES 1959-1967

YEAR	COUNTIES	CAUSES OF DEATH	PLACE OF RESIDENCE OF DECEASED	LENGTH OF RESIDENCE		OCCUPATION
				ARIZONA	TOWN	
1959	Gila & Pinal	Digestive Diseases	Yes	Yes	No	Yes
1961			↓	↓	↓	↓
1962	↓	↓	↓	↓	↓	↓
1964	↓	↓	↓	↓	↓	↓
1965	↓	↓	↓	↓	↓	↓
1967	↓	↓	↓	↓	↓	↓

Note: These data were available as hard copy. Since it involved only 144 certificates, it was not put on tape and could easily be manipulated by hand if the preliminary analysis indicated the need to examine digestive mortalities from this early time period.

MONTANA 1968-1975

YEAR	COUNTIES	CAUSES OF DEATH	PLACE OF RESIDENCE OF DECEASED	LENGTH OF RESIDENCE	
				MONTANA	TOWN
1968	All Montana Counties	All	Yes	No	No
1969		↓	↓	↓	↓
....		↓	↓	↓	↓
....	↓	↓	↓	↓	↓
1974	↓	↓	↓	↓	↓
1975	↓	↓	↓	↓	↓

UTAH 1965-1975

YEAR	COUNTIES	CAUSES OF DEATH	PLACE OF RESIDENCE OF DECEASED	LENGTH OF RESIDENCE	
				UTAH	TOWN
1965	All Utah Counties	All	No	No	No
1966		↓	↓	↓	↓
....		↓	↓	↓	↓
....	↓	↓	↓	↓	↓
1974	↓	↓	↓	↓	↓
1975	↓	↓	↓	↓	↓

b. Demographic Data Bases

Census data were used in the selection of comparison areas and in the determination of denominators for mortality rates. These data are from the fifth count of the 1970 U.S. Census of Population and were made available on computer tape by the Bureau of the Census. The data are grouped by enumeration districts, which are administrative divisions devised by the Bureau of the Census that respect boundaries of towns and contain a population of about 800 each. The vast majority of the data points used are based on a 15% sample and are subject to some chance variability due to sampling. Nevertheless, the variability can be estimated, and its importance is lessened by the aggregation of several enumeration districts into exposed and unexposed areas. Census tapes were obtained for the states of Arizona, Montana, and Utah.

In addition to the Census tapes, other demographic and background information was obtained from local chambers of commerce, banks, and other public affairs offices. Conversations with local individuals during site visits were also useful.

c. Air Pollution Data Bases

- *Arizona*
  - —Arizona Air Quality Reports 1969-1975
  - —Estimated Impact of Smelter Operations on SO_2 Concentrations in the Rocky Mountain Area
  - —SAROAD and NEDS data

- *Montana*
  - —Helena Valley Area Environmental Pollution Study (EPA)
  - —Montana Air Quality Bureau Reports
  - —CHESS study (EPA)
  - —SAROAD and NEDS data

- *Utah*
  - —Utah Ambient Air Pollution Reports
  - —A Summary of Air Pollution Source Emission Calculations for Utah
  - —SAROAD and NEDS data
  - —CHESS study (EPA)

d. *Industrial Data*

Data on the industry were obtained through standard textbooks, conversations with officials, brochures from the companies, and from reports prepared by EPA.

e. *Morbidity Data*

Due to the cost and effort involved in the acquisition of hospital morbidity data, it was used in only one of the studies in this program (bituminous coal mining). However, during our visit to Arizona, we interviewed the administrators of three hospitals* in order to obtain an overview of the community health problems and find out if there were any readily available reports or records that might be useful to our study.

Two of the hospitals visited were owned and operated by the industry. They provided useful background information, but said that they could not release any more specific information until the case for involvement of the smelters in community disease was more clear-cut. They referred to a very valid point: the finding of a high mortality rate in a county with a large industrial operation sometimes leads to a hasty indictment of the industry before all the facts are in. Since the company towns constitute only a small fraction of the total population of a county, it must first be established whether the deaths in the town itself are excessive.

Apart from the question of hospital cooperation, it was determined that any collection of morbidity data from these hospitals would be a difficult job. Records are not indexed by town of residence of the patient, so the name file would have to be examined card by card to identify patients from the towns of interest. Retrieval by residence is theoretically more feasible for those hospitals which subscribe to automated records systems such as the Professional Activity Study (PAS) and the Hospital Utilization Project (HUP), since the file can be searched on zip code. Unfortunately for retrospective studies such as ours, these systems are relatively new and the inclusion of zip code patient identifiers has only been in practice for the last few years.

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*Miami-Inspiration Hospital; Kennecott Copper Corporation Hospital; Pinal General Hospital.

D. *RESULTS*

The mortality rates for 34 disease groups for white females in the smelter towns were analyzed as described in Section C of this chapter. The rates for the diseases which were elevated in at least one of the three towns of Douglas, Anaconda, and Magna are shown in Table V-6. The supporting data for this set (SMRs, relative risk, χ^2 values, and control data) are given in Tables F-1 through F-3 in Appendix F. The mortality rates for all other diseases for these towns are included for reference in Appendix F, Tables F-15 through F-17.

Table V-6

ELEVATED MORTALITY RATES IN DOUGLAS, ANACONDA, AND MAGNA

ECI #	CAUSE OF DEATH DISEASE CATEGORIES[a]	AGE-ADJUSTED MORTALITY RATES[b]			
		Douglas, Arizona	Anaconda, Montana	Magna, Utah	National Averages
5	Cancer of the colon & rectum	33	56	33	N.A.
7	Cancer of the pancreas	5	39	0	12
10	Cancer of the genitourinary organs except bladder	63	45	54	45
12	Other cancers except leukemia	110	98	131	90
14	Blood, endocrine, nutritional & metabolic diseases	57	24	77	34
18	Cardiovascular diseases	382	443	379	384
19	Cerebrovascular diseases	79	133	70	84
20	Acute respiratory diseases	60	45	11	26
27	Diseases of liver, gall-bladder & pancreas	67	56	33	N.A.
31	Diseases of the skin & musculoskeletal system & ill-defined diseases	61	21	57	N.A.

[a] The specific diseases involved in these categories are given in Appendix G.

[b] Number of deaths per 100,000; white females; ages 35-74; 1968-1975 (SMRs, relative risks, and χ^2 are given in Appendix F). The age-adjusted mortality rate was calculated for deaths occurring in the 35-74 age group.

Underlined rates are significantly higher than at least one control area at, at least, the $p < 0.05$ level.

N.A. = National averages not available for these disease categories.

From Table V-6 we can make the following observations:

- No one disease is significantly elevated in all three smelter towns, although two diseases are elevated in two of the towns. Disease 12 (cancers of the glands and lymph nodes, secondary neoplasms, and benign neoplasms) are high in Douglas and Anaconda. Also elevated in Douglas and Anaconda is disease 27 (diseases of liver, gallbladder, and pancreas).
- Colon, rectum, and pancreas cancer are high in Anaconda.
- Genitourinary cancer, acute respiratory diseases, and skin/musculoskeletal disorders are high in Douglas.
- Cardiovascular diseases and metabolic/nutritional disorders are high in Magna.

Table V-7 shows the results of the analysis of combined disease groupings for the aggregate of six Arizona smelter towns; the actual number of deaths involved in these groupings is shown in Table F-4 in Appendix F. The mortality rates for the individual towns included in the aggregate are given in Appendix F, Tables F-15 through F-17.

Table V-7

MORTALITY RATIOS FOR ARIZONA SMELTER TOWN AGGREGATE

CAUSE OF DEATH (ECI #)	MORTALITY[a] RATIOS		$\frac{\text{Smelter Town Ratio}}{\text{Control Area Ratio}}$
	Smelter Towns[b]	Control Area[c]	
Organ cancers (2-11)	.0053	.0051	1.03
Other cancers except leukemia (12)	.0036	.0016	2.26
Neoplasms of the lymphatics (13)	.0015	.0008	1.88
Vascular diseases (17-19)	.0307	.0333	0.92
Respiratory diseases (20,21)	.0050	.00049	10
Digestive diseases (22-27)	.0037	.001	3.76
Genitourinary diseases (28,29)	.00125	.0007	1.79

[a] White females, all ages, 1968-1975.

[b] Mortality ratio = $\frac{\text{number of WF deaths in smelter towns}}{\text{sex-, race-specific population in smelter towns}}$

[c] Mortality ratio = $\frac{\text{number of WF deaths in control area}}{\text{sex-, race-specific population in control area}}$

From Table V-7 we see that, in the smelter town aggregate, acute respiratory diseases show the greatest elevation.

Our next step, after identifying the diseases which are elevated in the smelter areas, was to attempt to explain the excess mortality in terms of environmental pollution. The following sections describe the three disease-pollution hypotheses which resulted from the integration of environmental and mortality data. No environmental explanation for the remainder of the elevated diseases was found.

1. Sulfur Dioxide and Particulates

Data on the pre-emission control levels of five major smelter pollutants (sulfur dioxide, particulates, cadmium, lead, and arsenic) for the eight sites were collected and analyzed. Table V-8 summarizes the findings for sulfur dioxide and particulates for the sites. This table represents the condensation of a great deal of data from several sources. Tables F-5 through F-9 and Figure F-1 in Appendix F tabulate in detail the figures from which this summary was derived.

Table V-8

*SUMMARY OF SULFUR DIOXIDE AND PARTICULATE POLLUTION FOR ARIZONA, UTAH, AND MONTANA**

AREA	TIME PERIOD	SULFUR DIOXIDE[a] (μg/m^3)			PARTICULATES[b] (μg/m^3)		
		Range	Avg ±σ	Category	Range	Avg ±σ	Category
Arizona Aggregate	1969-1971	107-151	135 ± 24	High	111-141	122 ± 16	High
Magna, Utah	1969-1971	61-103	83 ± 20	Medium	55-70	63 ± 7	Low
Anaconda, Montana[c]	1965-1966	40-91	60 ± 17	Low			
	1961-1962				10-339	89[d]	Medium

*Detailed data for this summary is given in Appendix F, Tables F-5 through F-9 and Figure F-1.

[a] Annual standard: 50 μg/m^3. [b] Annual standard: 70 μg/m^3.

[c] Data for 1969-1971 not available. Data for the three three areas is comparable, insofar as it is all from the pre-emission control period.

[d] Standard deviation not available from data source.

Based on the available data and the existing standards for sulfur dioxide and particulates, the Arizona, Utah, and Montana sites fall into different exposure categories. The Arizona sites are high in both sulfur dioxide and particulates; Magna has an intermediate level of exposure from sulfur dioxide and is low in particulates. Anaconda is intermediate in particulates and low in sulfur dioxide. Table V-9 shows in greater detail the lengths of time that sulfur dioxide and particulates in Arizona were in excess. The range for sulfur dioxide was from 15 to 193 days per year in excess of the state's daily standard of 250 $\mu g/m^3$. Douglas exceeded the standard for 138 days. Ajo, Claypool, Douglas, and Hayden exceeded both the annual and the daily standard for particulates; Douglas and Hayden were by far the highest. Figure V-3 indicates that both Douglas and Hayden exceeded the maximum daily standard of 100 $\mu g/m^3$ 96% of the time in 1969; Ajo slightly exceeded the annual standard of 70 $\mu g/m^3$ in 1969.

Table V-9

DETAILED SULFUR DIOXIDE MONITORING FOR ARIZONA SITES ($\mu g/m^3$)

	AJO	CLAYPOOL	CLIFTON	DOUGLAS		HAYDEN	SAN MANUEL
				North	South		
Monitoring Period	3/19-4/21 7/7-12/31	2/7-12/31	10/1-12/31	11/13-12/31	4/2-12/31	1/17-12/31	5/22-12/31
Percent Data Recovery	77	80	84	51	63	60	77
Maximum Hourly Average	3,928	4,910	2,455	4,664	3,862	4,752	4,910
Percent of Hours > 850 $\mu g/m^3$ (State Hourly Standard)	1.5	2.9	0.9	2.0	3.6	12.5	1.4
Maximum Daily Average	1,105	565	417	908	515	3,849	908
Percent of Days > 250 $\mu g/m^3$ (State Daily Standard)	5.0	18.2	4.0	8.0	38.8	53.7	17.2
Annual Average	98	147	90	109	64	377	147

Source: Arizona State Department of Health, Division of Air Pollution Control, 1970.
State Annual Standard is 50. [2,860 $\mu g/m^3$ of SO_2 = 1 ppm (volume at 0°C, 1 atm.]

The analysis of the mortality rates showed that deaths from acute respiratory diseases among white females were excessive in Douglas and in the smelter town aggregate. On the basis of the available data, it thus appears that the excess mortality from acute respiratory diseases in Arizona is best explained by the effects of a combination of high particulate and high sulfur dioxide pollution. The evidence to support this relationship is summarized in Table V-10.

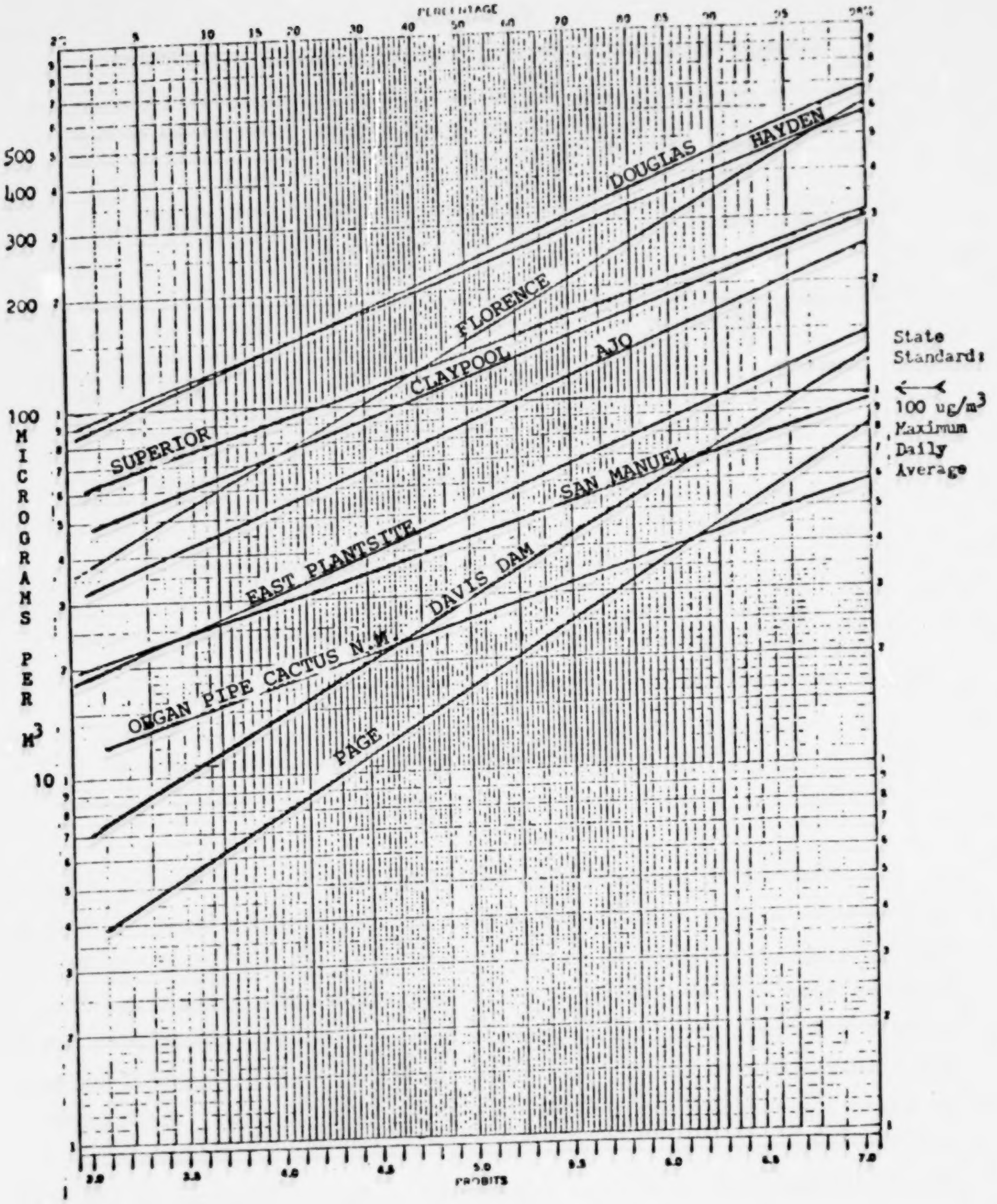

Figure V-3. Arizona: Percent of Time Concentrations of Particulates Less Than Stated Value in 1969 (from Arizona Department of Health, Division of Air Pollution Control, September 1970)

Table V-10

SUMMARY DATA FOR ACUTE RESPIRATORY DISEASES HYPOTHESIS

AREA	MORTALITY RATE FROM ACUTE RESPIRATORY DISEASE: WHITE FEMALES	SULFUR DIOXIDE EXPOSURE	PARTICULATES EXPOSURE
ARIZONA SMELTER AGGREGATE	EXCESSIVE 10-fold excess over control area.	HIGH Annual average is 135 $\mu g/m^3$; standard is 50. SO_2 levels > 250 $\mu g/m^3$ for 15 to 193 days in 1969.	HIGH Annual average is 122 $\mu g/m^3$; standard is 70.
DOUGLAS	EXCESSIVE 60[a]	HIGH SO_2 levels > 250 $\mu g/m^3$ for 138 days in 1969.	HIGH Exceeded maximum daily standard of 100 $\mu g/m^3$ 96% of the time in 1969.
ANACONDA	NOT EXCESSIVE 45[b]	LOW Annual average: 60.	MEDIUM Annual average: 89.
MAGNA	NOT EXCESSIVE 11[b]	MEDIUM Annual average: 83.	LOW Annual average: 63.

[a] Significantly higher than controls at $p < 0.05$.

[b] Not significantly different from controls.

PHYSIOLOGICAL DATA:

- Particulates and sulfur dioxide in combination have deleterious effects upon the respiratory system.
- Levels of sulfur dioxide > 240 $\mu g/m^3$ for more than one month are deleterious.
- Particulate levels of 70 $\mu g/m^3$ (annual) or 100 $\mu g/m^3$ (daily) are considered unhealthy.

The omission of corresponding pollution data for the control areas is unavoidable because such measurements have not been done on a routine basis for areas suitable as controls in studies of this type. In our conversations with the local pollution officials, the comment was frequently made that there is a definite need to establish monitoring networks in nonproblem areas which could serve as potential control sites in pollution-disease correlation. In the case of Arizona, for example, the only data available for even general comparison purposes were recently released by the Arizona Department of Health Services in its 1976 Air Quality Data for Arizona. The annual average data indicate that hazardous levels of SO_2 are not found in nonsmelter areas such as Tucson and Phoenix. Even in 1976 when pollution control was strictly enforced, it was evident that smelter towns still exceed nonsmelter towns in pollution from sulfur dioxide (see Table V-11). This rank order can reasonably be expected to have been more pronounced in the pre-1970 era focused on by this study. Hence, although the data in Table V-10 lack the rigorousness of a comparison of exposed and nonexposed pollution data, it is reasonable to assume that the SO_2 levels in the control areas were low enough to make the comparison implied in Table V-10 a valid one.

Table V-11

1976 ANNUAL AVERAGES FOR SULFUR DIOXIDE (μg/m^3)

Smelter Towns	
Ajo (5 sites)	16 ± 10
Douglas (10 sites)	34 ± 11
Hayden (6 sites)	103 ± 29
Miami (2 sites)	38 ± 24
Morenci (10 sites)	49 ± 52
San Manuel (5 sites)	44 ± 20
Nonsmelter Cities	
Phoenix (5 sites)	7 ± 0.5
Tucson (7 sites)	5 ± 2

Source: Arizona Department of Health Services. (Raw data for individual sites is given in Table F-18, Appendix F.)

2. *Heavy Metals*

Table V-12 summarizes the levels of cadmium, lead, arsenic, and fluoride in the Arizona and Montana smelter sites. The available data for Utah is in units which cannot be compared to that for Arizona and Montana. The records used to generate these summaries are detailed in Tables F-10 through F-13 in Appendix F. Since there is much less basic toxicological data (and consequently no environmental standards) for these elements than for SO_2, it is difficult to designate areas as "high" or "low." Furthermore, there has not been routine monitoring for heavy metals for the comparison areas as is the case with the sulfur dioxide analysis discussed above. With these limitations in mind, we can draw the following conclusions about heavy metal pollution in smelter towns.

Table V-12

SUMMARY OF HEAVY METAL POLLUTION FOR ARIZONA AND MONTANA

AREA	YEARS	POLLUTION	
		Atmospheric ($\mu g/m^3$) Average ±σ	Nonatmospheric
CADMIUM			
Arizona Smelter Towns	1969	.006 ± .005	
Arizona Towns Without Smelters	1969	.004 ± .005	
Anaconda	1971-72	.03 ± .02	3.6 ppm (soil)
LEAD			
Arizona Smelter Towns	1969	1.2 ± 1.2	
Arizona Towns Without Smelters	1969	0.18 ± 0.2	
Anaconda	1961-62	0.54	
ARSENIC			
Arizona Smelter Towns	1969	.0065 ± .0027	
Arizona Towns Without Smelters	1969	.0042 ± .0042	
Anaconda	1961-62	0.45	33 ppm (soil)
FLUORIDE			
Anaconda (West)	1965		12.6 ppm (grass)

(1) Contamination by cadmium is less of a problem in Arizona as a whole than it is in Anaconda. The levels in Anaconda approach the upper limit of those measured in a nationwide survey of nonurban areas; these values were 0.0004 to 0.026 $\mu g/m^3$. Based on a sample taken in 1977, levels of soil cadmium in a residential street adjacent to the plant are at least three times average soil levels. The town of Hayden in Arizona has a level from 10 to 100 times higher than the other five Arizona smelter towns.

(2) Contamination by arsenic is much less of a problem in all Arizona smelter towns than in Anaconda. Anaconda has the highest arsenic

level in Montana and is also higher than New York City and Chicago. Based on a sample taken in 1977, levels of soil arsenic are about four times higher than average soil levels.

(3) Lead levels in the Arizona smelter towns were high in comparison to the nationwide survey results of 0.002 to 0.15 $\mu g/m^3$ in nonurban areas. Anaconda is lower than Arizona, but still above the nationwide levels.

Furthermore, fluoride, although not emitted from copper smelters, is present in high levels in West Anaconda.

Table F-14 in Appendix F shows the heavy metal body burden data from two studies. One is from Anaconda and Bozeman done on hair in 1969; the other was done with hair, urine, and blood samples from children in 1975. The body burden data support the conclusion that individuals who live in smelter areas take up and retain lead, cadmium, and arsenic and have bodily levels significantly different from individuals living in nonsmelter towns. The body burden data do not appear to follow the same rank order as the environmental pollution levels, although such a comparison may not be warranted due to the difference in time periods of the two data sets and the use of children as subjects in the Baker study [18].

When compared to mortality rates from the control areas, death rates from cancers of the colon and pancreas and from cerebrovascular diseases were elevated in Anaconda but not in Douglas. The excessive rates from the cancers in Anaconda may be explained by the presence of high levels of suspected carcinogens (cadmium, arsenic, fluoride) in that area. The excessive rate from cerebrosvascular disease may be accounted for by the high cadmium levels in Anaconda. The evidence for this hypothesis is summarized in Table V-13.

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[18] Baker, E. L., C. G. Hayes, P. J. Landrigan, J. L. Handke, R. T. Leger, W. J. Housworth, and J. M. Harrington, A nationwide survey of heavy metal absorption in children living near primary copper, lead and zinc smelters. *Am. J. Epidemiol 106*(4):261-273, 1977.

Table V-13

SUMMARY DATA FOR HEAVY METAL HYPOTHESIS

AREA	CAUSE OF DEATH	AGE-ADJUSTED MORTALITY RATE: WF	CADMIUM ($\mu g/m^3$)	ARSENIC ($\mu g/m^3$)	FLUORIDE (ppm, g)
Anaconda	Cancer of colon	56	.03	0.45	12.6
	Cancer of pancreas	39			
	Cerebrovascular disease	133			
Douglas	Cancer of colon	33	.011	.004	NA
	Cancer of pancreas	5			
	Cerebrovascular disease	79			

Underscored rates are significantly higher than control values.

NA = Data not available.

National averages (WF) (mortality/100,000): Cancer of colon ≤ 26
Cancer of pancreas 39
Cerebrovascular disease 84

PHYSIOLOGICAL DATA:

- Recent toxicological and epidemiological evidence indicate that cadmium, arsenic, and fluoride are all implicated as possible carcinogens. Cadmium has been linked with vascular and hypertensive disease as well. (See Section B above.)

As pointed out above, because there is insufficient heavy metal data for the comparison areas and a lack of environmental standards, we can only make a rank order comparison. Thus, the available data point to the fact that in Anaconda there are high levels of cancer of the colon and pancreas as well as cerebrovascular disease which occur in conjunction with elevated environmental levels of pollutants that have been associated with these diseases. In Douglas, rates for the diseases are not elevated with respect to control values, and the levels of suspect pollutants are low.

3. *Digestive Diseases*

Diseases of the liver, gallbladder, and pancreas (disease 27) are elevated in Douglas, Anaconda, and the Arizona smelter town aggregate. A breakdown of individual diseases included in this grouping is given in Table V-14. From these data we see that 70 percent (24 out of 34) of the deaths are from cirrhosis of the liver; 38 percent (9 out of 24) of the cirrhosis cases were listed on the death certificate as cirrhosis due to alcoholism. In Arizona, it appears that most of the deaths in this category are due to alcoholic cirrhosis. The diagnosis of "unspecified cirrhosis" may indicate that the disease is not related to alcohol consumption or it may reflect a local tendency to omit mention of alcoholism on death certificates in deference to family sensitivity on the issue, a practice which has been reported to occur. Although lead, cadmium, and arsenic have all been described as either accumulating in and/or actually damaging the liver, the evidence is not sufficient to suggest a role for these metals in the aggravation of alcoholic cirrhosis.

Table V-13

DISEASES OF LIVER, GALLBLADDER AND PANCREAS: NUMBER OF DEATHS (WHITE FEMALES, 1968-1975)

	DISEASES OF LIVER, GALLBLADDER & PANCREAS	NUMBER OF DEATHS											
		Miami	Hayden	Ajo	Douglas	San Manuel	Morenci	Anaconda	Magna	Row Totals	All of Arizona	All of Montana	All of Utah
Cirrhosis	Alcoholic cirrhosis of liver	0	0	2	3	0	2	0	2	9	471	58	105
	Other specified cirrhosis of liver (without alcoholism)	0	0	0	1	0	0	2	1	4	80	31	35
	Unspecified cirrhosis of liver (without alcoholism)	0	0	0	3	0	0	6	2	11	128	99	96
Non-Cirrhosis	Acute & subacute necrosis of liver	0	0	0	1	0	0	1	0	2	16	30	31
	Suppurative hepatitis & liver abscess	0	0	0	0	0	0	0	0	0	2	10	14
	Other diseases of liver	0	0	0	0	0	0	0	0	0	66	15	22
	Cholelithiasis	0	0	0	0	0	0	0	0	0	34	10	16
	Cholecystitis & cholangitis	0	0	0	2	0	0	0	0	2	31	10	14
	Other diseases of gallbladder & biliary ducts	0	0	0	1	0	0	0	0	1	15	10	14
	Diseases of pancreas	0	0	0	2	0	0	2	1	5	65	26	39
	Column Totals	0	0	2	13	0	2	11	6	34	908	299	386

4. *Factor Analysis*

Specific data on the industry, environment, and demographics were assembled for 11 Arizona towns in the proximity of copper smelters. The towns range from 0.3 to 9 miles from smelters. Table V-15 lists the towns and available data. These data, together with disease-specific mortality rates in white females for the towns, were subjected to an iterative factor analysis to attempt to find the commonalities among the data, i.e., to identify groups of variables that are associated with common factors. The purpose of the analysis was to explore the patterns in the data to see if certain diseases appear to be associated with specific variables, either socioeconomic, industrial, or environmental in nature. Patterns and associations in the data involving disease variables were then tested for strength of association by means of a multiple regression analysis in which the disease mortality rate was taken to be the dependent variable.

This analysis resulted in only three strong associations. These are shown in the following table.

DISEASE	ASSOCIATED VARIABLES	STEPWISE MULTIPLE REGRESSION COEFFICIENT (R)	$(\Delta R)^2$
Acute Respiratory Disease (pneumonia, etc.)	• Ambient SO_2 concentration	0.84	0.70
	• Age of smelter	0.88	0.07
Digestive Disease (primarily liver cirrhosis)	• Percent population white	0.44	0.02
	• Tons per day of concentrates	0.64	0.21
	• Percent not completing high school	0.91	0.43
Cancer of Genito-urinary Organs	• Number of persons per room	0.74	0.55
	• Tons per day of concentrates	0.81	0.11

The clearest association is that between respiratory disease and ambient SO_2 concentrations. To a much lesser degree, the disease is associated with the age of the smelter. These two variables together

Table V-15

DEMOGRAPHIC, ENVIRONMENTAL AND INDUSTRIAL DATA FOR ARIZONA TOWNS IN VICINITY OF COPPER SMELTERS

	ARIZONA TOWNS IN VICINITY OF COPPER SMELTERS										
	Hayden	San Manuel	Miami	Superior	Claypool	Globe	Kearny	Winkelman	Ajo	Morenci	Douglas
Specific Population[a]	209	482	638	790	453	2,017	448	150	979	663	2,400
Median Age	21	18	23	23	30	30	23	20	30	30	23
Median Income	9,500	11,000	7,500	8,500	7,500	8,500	11,000	9,500	9,500	9,500	7,500
% in Same House[b]	70	32	63	61	70	58	48	48	42	51	58
% White[c]	100	98	99	98	95	98	96	100	90	97	98
% with <1 person per room	71	77	83	81	84	90	93	81	83	88	84
% Completed High School	42	63	41	37	45	49	66	38	50	52	43
% Female	51	49	51	50	51	51	49	50	51	51	53
% Native-Born Parents	69	85	71	72	85	85	85	72	72	84	43
Age of Smelter (years)	60	16	57	47	57	57	60	60	25	33	65
Distance to Nearest Smelter (miles)	0.3	1	0.85	0.5	0.85	2.5	9	0.6	0.5	0.5	0.7
Ambient Arsenic (μg/m^3)	--[e]	.01 ±.002	--	.01 ±.01	.01 ±.003	--	--	.004 ±.002	.004 ±.002	.0045 ±.003	.02 ±.02
Ambient Lead (μg/m^3)	--	0.15 ±.07	--	0.58 ±0.10	0.93 ±0.31	--	--	3.0 ±1.4	0.27 ±0.21	1.2 ±1.4	1.13 ±0.69
Ambient Cadmium (μg/m^3)	.016	.003 ±001	.01 ±.01	.01 ±.01	.01 ±.01	--	--	.004	.002 ±.018	.005 ±.001	.002 ±.002
Ambient SO_2 (μg/m^3)	367±87	102±40	80±47	158±132	80±47	--	--	35±17	95±67	115±20	80±40
Particulates[d] (μg/m^3)	224	--	--	142	--	--	--	--	78	--	--
Concentrates (tons/day)	2,000	1,700	840	--	840	840	2,000	2,000	680	2,113	2,260
Blister Copper (tons/day)	366	310	300	--	300	300	366	366	197	470	365
Stack Height (feet)	300	515	275	--	275	275	300	300	360	600	556
SO_2 Concentration (ppm)	5,900	15,000	2,500	--	2,500	2,500	5,900	5,900	7,000	14,000	15,200
Particulates Emitted (tons/day)	1.5	1.79	--	--	--	--	1.5	1.5	0.7	14.7	9
SO_2 Emitted (tons/day)	290	313	78	--	78	78	290	290	380	526	492

[a] All demographics based on 1970 Census.

[b] Since last census.

[c] Nonwhite includes Indians and blacks.

[d] Insufficient data for use in factor analysis.

[e] -- = data missing; row mean used in factor analysis.

are strongly correlated with the disease mortality rate in females (R = 0.88). Further, no appreciable correlation was found between respiratory disease and the socioeconomic variables. This result provides evidence that SO_2 is the principal etiologic agent contributing to elevated respiratory disease mortalities.

The other two associations found appear to be principally socioeconomic relationships. In both cases, the only industry-related variable is the production variable of tons per day of concentrates. It is not clear what this variable represents. It could be a surrogate measure of some emission or effluent which was not present in the data, or it could represent more of a socioeconomic measure related to total number of employees and total income generated by smelter workers.

E. CONCLUSIONS AND RECOMMENDATIONS

1. Conclusions

- Three hypotheses resulted from this study which link a specific industry emission to an excess mortality pattern. They are discussed in detail in Section D.

- The regression analysis, using county-level data, successfully pointed out elevated mortalities which proved also to be high when examined on a town-by-town basis. In the case of respiratory disease, the original regression category was very broad. The present study narrowed down the diseases involved considerably. In the case of digestive diseases, the most likely cause is not industry pollution, but rather excess alcohol consumption. The usefulness of the regression technique to objectively detect community health problems is limited by quantitative factors such as the ratio of town-to-county populations, prevalence of the disease, and influence of socioeconomic factors.

2. *Recommendations*

- The regression methodology can be made more specific by the use of mortality figures from the towns actually at risk from a particular industrial pollutant, rather than the rates for the entire county which may considerably dilute the town effect. Screening for town-level effects can be accomplished by the approach used here, i.e., use of data obtained from state registries. It would be highly desirable if all states included in their mortality certificates and tapes the length of the decedent's residence in the town and his or her occupation. This would be a valuable epidemiological tool.

- There is a need for more environmental data for control, non-industrial areas.

- The relationship between ambient pollution levels and individual exposure needs further evaluation. It would be useful to perform pilot studies in towns such as those described here, using personal monitors on groups of residents to more precisely determine their exposure.

- The work described here represents a study of about half of the primary copper smelters in the United States. Three specific industry-disease associations have been identified. It would be useful to determine if these associations occur in the exposed populations of the remaining smelters in the country.

Chapter VI

STEEL MANUFACTURING CASE STUDY

A. *INTRODUCTION AND SUMMARY*

1. *Study Objectives*

The objective of this case study is to explore the relationship between the steel industry and community disease, in particular, digestive cancers and cardiovascular diseases. The correlations between these diseases and the steel industry were generated by the multiple regression analysis of county industry and mortality data. This case study is a field test to attempt to ascertain the nature of the relationship between the industry and the diseases. The results of this investigation are to assist in the evaluation of the effectiveness of the multiple regression analysis for identifying true associations between industry and community disease. Thus, the ultimate purpose of this case study is to assist in the development of methodology for identifying sources of harmful substances affecting the health of the community.

The regression analysis correlated the presence of the steel industry (SIC 3312) in counties with elevated mortalities from digestive neoplasms. Testing this correlation involved finding a plausible cause-and-effect hypothesis at the sites studied. We chose to study Johnstown, Pennsylvania, as our hypothesis development site and Provo, Utah, for hypothesis test. After determining which steel processes (and therefore which emissions) were present at the selected sites, which emissions had a plausible etiology for the relevant diseases, and what the ambient air levels of these emissions were, we were able to delineate a population potentially at risk. We were then able to analyze mortality data in terms of potentially exposed and unexposed areas and determine significant elevations in disease mortality rates and to associate them with specific etiologic agents.

Several methodologies were developed in the course of these analyses to utilize the data available. Transport analyses were devised by our staff as well as by EPA's Environmental Photographic Interpretation Complex (EPIC) to characterize pollution patterns and populations at risk. EPIC data were used to get a historical perspective on changes in both industrial activity and population by comparing aerial photographs from as far back as 1939 to the present. The availability of cancer registry data made it possible to analyze incidence rates and other factors besides mortality. Finally, several statistical analyses were developed and used, depending on data type and availability, to determine significant differences between test and control values.

2. *Study Approach*

The field study was conducted at two sites. The first site constituted the hypothesis development site and the second, the hypothesis test site. Both sites were chosen using the basic assumptions that the study county should be no larger than 200,000, should have a high industrial index for the SIC code 3312, and should contain no other industry that might produce confounding emissions. These conditions being met, the hypothesis development site (Johnstown, Pennsylvania) was chosen because that county showed elevated mortality rates for the diseases of interest. The hypothesis test site (Provo, Utah), on the other hand, was chosen using only the basic assumptions. Of course, in both cases data availability was an important factor in choosing the site.

For each site and its control, we obtained the following data:

- Mortality and census data—used to calculate age-adjusted mortality rates for each site.
- Air quality data—to determine population exposure.
- Site-specific industry data—production figures and emission control data to determine historical exposure as well as emissions present.

The data for Johnstown were analyzed, plausible etiologies developed in light of those causes of death found elevated, and hypotheses developed. The Provo data were analyzed in terms of the hypotheses developed at Johnstown. Possible explanations for any differences found in the two sites were explored.

3. *Key Findings*

The hypotheses developed in Johnstown are summarized below:

In both males and females—

- Various malignant neoplasms (ICDA codes* 140-151 oral cancers, 158-163 digestive cancers, 170-174 breast cancers, 190-239 central nervous system cancers and leukemias) caused by high-level exposure to organic emissions, notably BaP.
- Cardiovascular diseases (ICDA codes 400-458) caused by exposure to high cadmium levels.
- Respiratory diseases (ICDA codes 460-519) caused by high exposure to particulates, SO_2, and trace metals.

In males only (possibly occupational)—

- Endocrine, metabolic, and blood diseases (ICDA codes 240-289) due to exposure to trace metals.
- Liver diseases (ICDA codes 570-577) due to exposure to organics and trace metals.

In females only—

- Urinary diseases (ICDA codes 580-599) from trace metal exposure.

We then tested these hypotheses using the Provo data. The following hypotheses were well supported by the Provo data:

In both males and females—

- Various malignant neoplasms (ICDA codes 170-174 breast cancers, 190-199 central nervous system cancers, 210-239 benign neoplasms of the central nervous system) causes by exposure to organic emissions such as BaP.

In males only (possibly occupational)—

- Endocrine, metabolic, and blood diseases (ICDA codes 240-289) due to trace metal exposure.

Several other hypotheses were to a lesser degree supported by the Provo data, i.e., mortality rates were elevated but not significantly.

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*Note: All ICDA codes refer to the 8th Revision.

The causes of death in these cases included respiratory cancers, where in Utah the mortality rates were elevated only in males, and there not significantly. The cardiovascular diseases, elevated only in Utah women, were not significantly higher. While leukemias were significantly higher in males in Johnstown, they were only slightly elevated in males in Utah. The same was true for respiratory diseases in females.

By comparing the pollutant levels in Provo to those in Johnstown, the lower death rates in Provo can be readily explained. The pollutant levels at the Utah sites are roughly an order of magnitude lower than at Johnstown. Thus, assuming the Johnstown hypotheses are valid, there is a logical explanation why the mortality rates for some of the diseases were not significantly elevated in Utah. This rationale for the Utah results does not "prove" that the Johnstown causal hypotheses were correct—it simply provides a reason why the Utah results may not "disprove" the Johnstown results.

4. Conclusions and Recommendations

Some of the hypotheses developed at Johnstown withstood the test at Utah, notably malignant neoplasms caused by exposure to BaP. The reason that other hypotheses developed at Johnstown did not withstand the test at Utah could have been due to the much lower exposure levels at Provo.

Before definite conclusions can be drawn, more detailed studies must be performed to determine the effect of such variables as occupation, tobacco and alcohol consumption, and duration of residence on the mortaility rate patterns. Also, more detailed air quality data should be obtained, especially for control sites.

B. BACKGROUND

1. Basis for Selecting Associations

Potential industry-disease associations identified from the regression data were examined statistically to determine those likely to be causally related. The 10 counties having the highest industrial index for steel (SIC code P047) were aggregated and the mean sex-specific age-adjusted mortality rates for the specific diseases indicated by the regression were calculated. These were compared to the mean United States mortality rates for the diseases. The results of this comparison are found in Table VI-1.

Table VI-1

AGGREGATED MORTALITY RATES—TOP 10 INDUSTRIAL COUNTIES SIC CODE P047 COMPARED TO U.S. MEAN

Disease	Aggregated Mean Mortality Rates		Number of Sigma from U.S. Means	
	WM	WF	WM	WF
Cardiovascular Disease	1,481	695	+8.5	+11.5
Ischemic Heart Disease	701	298	+5.3	+7.5
Myocardial Infarction	542	198	+11.8	+9.6
Digestive Neoplasms	110	69	+5.9	+2.1

Since many of these counties were highly industrialized, in an effort to exclude effects from other polluting sources or "urbanization" we also examined eight uncluttered counties with high industrial indices for SIC code 3312 (a component of P047). The results of this analysis are shown in Table VI-2.

Table VI-2

AGGREGATED MORTALITY RATES—UNCLUTTERED COUNTIES
SIC CODE 3312 COMPARED TO U.S. MEAN

Disease	Aggregated Mean Mortality Rates		Number of Sigma from U.S. Means	
	WM	WF	WM	WF
Cardiovascular Disease	1,435	685	+4.6	+9.4
Ischemic Heart Disease	677	292	+2.4	+5.9
Myocardial Infarction	519	193	+8.0	+7.8
Digestive Neoplasms	112	69	+5.9	+2.0

As the tabled data shows, the rates for counties containing 3312 were significantly higher than the U.S. mean, so a further analysis was done to compare these rates to a similar mean rate for an aggregate of matched control counties not containing the industry (see Table VI-3). The only association in which the mortality rates for the industrial counties were significantly higher than the U.S. mean and the mean for the aggregated control counties was that for digestive neoplasms. This association was therefore selected for further study.

Table VI-3

AGGREGATED MORTALITY RATES—UNCLUTTERED COUNTIES
SIC CODE 3312 COMPARED TO MATCHED CONTROL COUNTIES

Disease	Number of Sigma	
	WM	WF
Cardiovascular Disease	+2.4	-3.2
Ischemic Heart Disease	+1.4	-2.8
Myocardial Infarction	-0.6	-1.3
Digestive Neoplasms	+5.3	+3.2

2. *Description of Steel Industry*

From iron ore, scrap metal, oxygen, coal and limestone, the steel industry produces hundreds of different steels, each having unique physical properties. Basically, iron ore is converted into iron and the content of other materials in the iron is suitably adjusted by the removal of some materials and the addition of others. Reaction vessels hold up to 300 tons of material and their chemical contents are controlled to less than one percent.

The operation of making steel from iron ore with coal and oxygen is shown in Figure VI-1. Initially, cleaned and sized coal is reduced by heating in coke ovens to coke (almost pure carbon). Next, coke, limestone and iron ore are mixed in a blast furnace with enough O_2 to burn the coke. This reaction releases enough heat to reduce the ore to molten iron. The next step refines the iron to steel by oxidation in one of three types of furnaces: open hearth, basic oxygen, or electric arc. This process creates the steel alloys with their special properties. Usually, selectively separated scrap and other materials are added to the iron in the furnace, while recarbonizing materials are added to the molten steel as it leaves the furnace.

The sinter plant is basically a recycling process. Small iron-bearing fines are collected from the steel plant and agglomerated to a size suitable for blast furnace feed. The fines cannot be directly fed to the furnace, because the high rate of gas flow through the furnace would carry them out.

A wide range of pollutants are generated by the diverse processes described. Water is used in a steel plant to cool hot steel, to clean airstreams, to cool hot coke, etc. In the actual steelmaking process, water comes in direct contact with coke in the quenching process, causing hydrocarbons to be carried into the wastewater. The furnace off gases carry particulate matter which enters the plant's wastewater when water is used to scrub the gases. Consequently, the same pollutants in air emissions are also in wastewater effluents when water scrubbers are used.

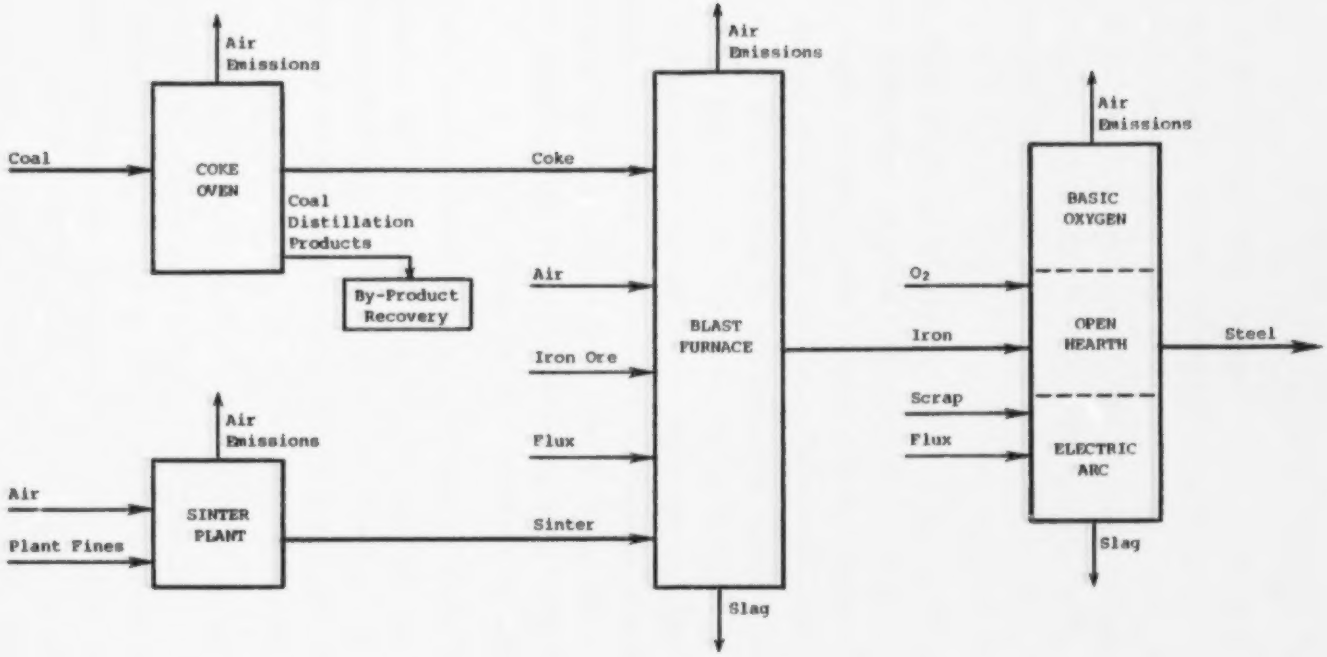

Figure VI-1. Steel Production

A broad range of pollutants from various sources are emitted into the atmosphere: hydrocarbons and oxides, gases and particulates, fugitive and stack emissions. Coke ovens reduce the complex hydrocarbons that make up coal, resulting in a large list of known and potentially toxic emissions, both particulate and gaseous. The sintering operation emits particulates, but they are hard to qualify due to the diverse nature of the feed. In blast furnaces, impurities are separated from iron ore that is reduced, creating gaseous and particulate emissions. Steel furnaces generate large amounts of respirable-sized particulates which are generally oxides.

a. *Coke Ovens*

Coke has been produced in beehive and in by-product, enclosed slot-type ovens. The beehive oven does not recover the by-products of destructive distillation of coal, while the enclosed slot-type does. About 99% of all metallurgical coke today is produced by the enclosed slot-type ovens. The beehive oven allows limited amounts of air into the chamber to burn with the volatile products distilled from the coal. The burning matter generates heat for further distillation. The enclosed slot-type is heated by external combustion of fuel gases around the oven walls.

A by-product coke oven is typically made of silica brick and is rectangular in shape (approximately 1.5' x 20' x 40'). Ovens are arranged side by side and are called batteries; as many as 100 ovens may be in a battery. Gases burning in flues in the walls between adjacent ovens, each of which holds 16 to 20 tons of coal, heat the coal up to 1100°F. Coking time varies from 16 to 24 hours, depending on oven size, coal quality, degree of coking achieved, etc.

Special cars (lorries) filled with coal, riding on rails on the roof of the battery, charge each oven with coal through holes (ports) in the top of the oven. The lorry cars discharge from the bottom and carry a measured weight of coal. A collection system, which runs the length of the battery, collects the gases and volatile matter distilled from the coal during the coking process under suction through pipes in the top of each oven. At the end of the coking process, the doors at each end of the oven are removed and a ram from a pushing machine which travels the length of the battery pushes the coke into a quenching car. The car carries the red-hot coke to a quenching tower where it is cooled by water sprays.

The gases and volatile materials (by-products) collected during the coking process may be recovered by various processes including tar decanting, recovery of phenol, distillation and absorption of ammonia, crystallization of ammonium sulfate, and fractionation, recovery and refining of light oils. The size of the plant and economics determine the extent of the recovery process employed; often the material is used as fuel inside the plant.

Emissions from the coking process are produced during coking, pushing and quenching (see Table VI-4). Their composition is varied depending on the coal used, degree of coking, degree of by-product recovery, etc. These emissions and their status with respect to health are further elucidated in Table VI-5. The particle size distribution is shown in Table VI-6.

Table VI-4. EMISSION FACTORS FOR COKING PROCESSES

Pollutant	kg/metric ton of Coal Charged	
	Coking Cycle	Discharging
Particulate	0.05	0.3
CO	0.3	0.035
Hydrocarbon[a]	0.75	0.1
NO_2	0.005	--
Ammonia	0.03	0.05

[a] Expressed as methane.

Table VI-5. *HAZARDOUS CHEMICALS PRESENT IN EMISSIONS FROM COKE PRODUCTION*

Known Hazardous Chemicals		Suspected Hazardous Chemicals	
Chemical Component	Class	Chemical Component	Class
α & β naphthyl amine 4-aminobiphenyl	amines	benzoic acid hydroxybenzoic acid hydrochloric acid sulfuric acid	acids and anhydrides
carbon monoxide	combustion gases	ammonia aniline methylanilines	amines
pyridine	heterocyclics	ammonium sulfate	inorganic salts
benzene toluene xylene	hydrocarbons	formaldehyde acetaldehyde paraldehyde	carbonyl compounds
phenol o-,m-,p-cresol	phenols	methylchrysenes benzo(a)anthracene dimethylbenzanthracene	polynuclear
pyrene chrysene benzo(a)pyrene benzo(e)pyrene dibenzo(a,h)anthracene dibenzo(a,g)fluorene	polynuclear	methyl mercaptan ethyl mercaptan	sulfur compounds
nickel carbonyl	organometallics	antimony beryllium silver vanadium	trace elements
thiophenes	sulfur compounds	ammonium thiocyanate	cyanides
selenium arsenic barium cadmium lead mercury	trace elements		
tar soot	fine particulates		
hydrogen cyanide ammonium cyanide	cyanides		

Table VI-6

PARTICLE SIZE DISTRIBUTION FOR A COKE OVEN EMISSION SAMPLE

Size, microns	% by Weight of Total Sample Collected on Plates
13.5	31.3
8.6	27.7
5.6	12.3
4.0	9.1
2.5	7.3
1.3	7.3
0.8	3.8
0.5	1.0

b. Sintering

The sintering process agglomerates fine iron-containing particles into a material suitable for charging into the blast furnace. Particles used in the process include fine ore, blast furnace flue dust, mill scale, turnings, coke fines, limestone fines, and other miscellaneous fines from the plant. They are placed on a grate and heated from above with a downdraft. At 2300 to 2700°F the fines agglomerate. The end process crushes, screens, and sizes the hot sinter.

Due to the material being handled—fine dust particles—sintering generates a large amount of air pollution. Handling generates fugitive emissions, and hydrocarbon fumes may be evolved when oily scrap is used. Also dependent on the feed material is the sulfur content of the gas stream which may reach 2,000 ppm. Particulate emissions are estimated to be 22 lb/ton of sinter. Table VI-7 describes the particle size distribution.

Controls on the sinter process include cyclones, electrostatic precipitators, baghouses, and scrubbers. Typically, a dry cyclone by itself will reduce emissions to about 2 lb/ton of sinter; while a dry cyclone in series with an electrostatic precipitator further reduces emissions to about 1 lb/ton of sinter.

Table VI-7. SINTERING EMISSIONS

Particle Size Distribution	
Size, microns	Percent Less Than By Weight
44	98
30	93
20	87
10	72
5	25

c. *Blast Furnaces*

A blast furnace is roughly cylindrical in shape and often measures 100 feet high and 20 feet in inside diameter. Overall height including the superstructure is often 150 feet. The structure has a steel shell lined with heat-resistant brick. Operating parameters range from 10 to 30 psi and from 400°F at the top of the furnace to over 3000°F at the bottom.

Generally, iron ore, coke, and flux are charged at the top of the furnace and hot air at the bottom. The air flows countercurrent to the falling charge. Carefully weighed coke, flux (limestone and others), and iron ore (including sinter pellets, mill scale, and iron or steel scrap) are carried to the top of the furnace in skip cars and charged to the furnace through a lock hopper. Air is compressed, then passed through a preheating vessel(cleaned furnace off-gas is used to heat the vessel), and introduced into the bottom of the furnace above the molten iron and slag. Oil or coal sludge is often injected along with the air.

As the coke descends through the furnace, it is preheated and finally burned. The heat of the reaction, combined with the hot air, reduces the iron ore (usually Fe_2O_3, Fe_3O_4, $FeCO_3$, and FeS) to Fe and then to molten iron. Fluxes combine with impurities to form a molten material, slag. Slag is lighter than the molten iron and floats on it at the bottom of the furnace.

Gases exit from the top of the furnace while the iron and the slag are periodically drawn off. The slag is usually handled in one of two ways: the air-cooling method pours the slag into dry pits in the ground to become one large solid; the other process rapidly cools the slag with high-pressure water streams and results in fine, brown sand-like particles. The molten iron is transferred by railcar to molds for pig iron, or to a holding vessel, or directly to a steel furnace. Gases exit at 400°F at a rate of 5 tons of gases/ton of iron. Table VI-8 describes the off-gas composition. An estimate of particle loading in the gas is 200 lb/ton of iron before control. Particulate composition is described in Table VI-9. Typically, particulates have been cleaned from the gas stream, because the hot steam is used to preheat the air entering the furnace—as well as in other processes involved in steelmaking—and particulates would clog the heat exchanger. Common controls on the gas stream include dry cyclones, web scrubbers, and electrostatic precipitators.

Table VI-8. BLAST FURNACE GAS COMPOSITION

Gas	Percent of Total Gases Emitted
CO_2	12.8
O_2	<0.1
CO	26.5
H_2	3.5
CH_4	0.2
N_2	56.9
H_2O	<0.1

Table VI-9

CHEMICAL COMPOSITION OF DRY, BLAST-FURNACE FLUE DUST

Component	Weight Percent (range)
Iron	36.5 - 50.3
Ferrous oxide	N.A.
Silicon dioxide	8.9 - 13.4
Aluminum oxide	2.2 - 5.3
Magnesium oxide	0.9 - 1.6
Calcium oxide	3.8 - 4.5
Sodium oxide	N.A.
Potassium oxide	N.A.
Zinc oxide	N.A.
Phosphorus	0.1 - 0.2
Sulfur	0.2 - 0.4
Manganese	0.5 - 0.9
Carbon	3.7 - 13.9

N.A. = not available.

d. *Open Hearth*

An open-hearth furnace is a shallow, rectangular refractory basin, which typically measures 30' x 90' and is lined with refractory material. Often several furnaces are operated in a line. Water-cooled doors are in the front, fuel jets are in opposite sides, an oxygen lance may be in the ceiling, and the taphole is in the lower back of the furnace.

Blast furnace iron, steel scrap, or equal portions of both are charged into the furnace in addition to a flux via the doors. Initially, flux (usually limestone) and scrap are dumped through the doors by the charging machine. Oil, tar, gas, etc., are burned from either end of the furnace. When the charge is partially melted, molten iron is poured into the furnace. Oxygen lances have been incorporated in newer furnaces and retrofitted onto many older ones for faster heating times. As the charge heats, carbon, manganese, phosphorus, sulfur, and silicon are removed from the iron, and they make up the slag along with the limestone which floats on the molten steel. When the steel has reached a predetermined carbon content at about 2900°F, the furnace is tapped. A large ladle is placed under the taphole and the plug in the taphole is blown out. As the steel flows into the ladle, other materials can be added to create the desired steel. Because the initial charge was carefully measured, the ladle fills with steel and the slag overflows into a smaller ladle alongside.

The flue gases pass through a heat exchanger which preheats the air needed for combustion ; its particle loading is described in Figure VI-2 and Table VI-10. Waste gases are composed of air, carbon dioxide, water vapor, sulfur oxides, nitrogen oxides, zinc oxides, and fluorides. The zinc oxide quantities are dependent on the use of galvanized steel scrap. Fluorides can be either gaseous or particulates and are generated when fluorspar (CaF_2) is used as a flux. Emission controls include electrostatic precipitators, venturi scrubbers, and baghouses, with the precipitator the device of choice.

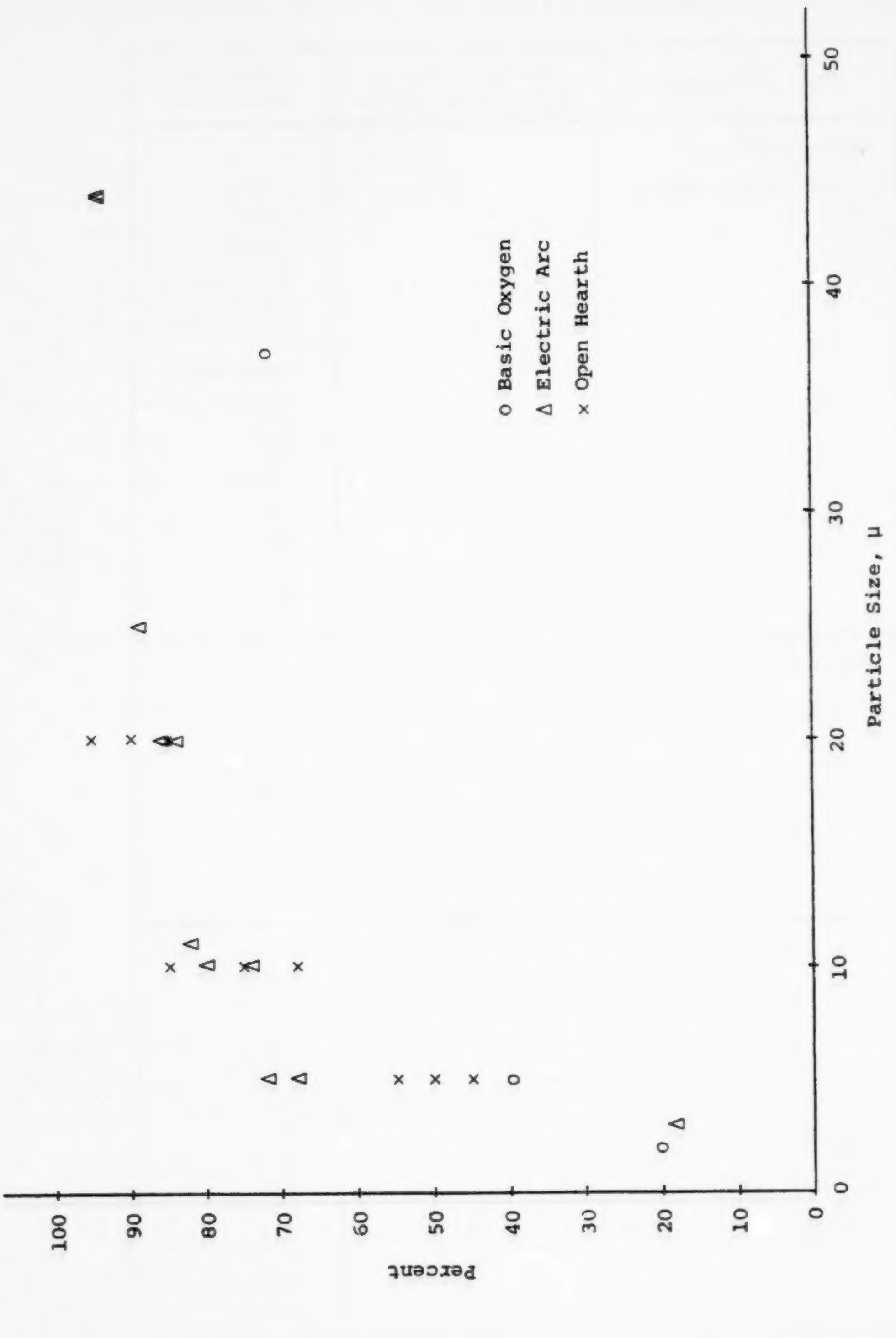

Figure VI-2. Steel Furnaces: Particle Sizes by Percent by Weight Less Than Stated Sizes

Table VI-10. STEEL FURNACE PARTICULATE EMISSIONS, UNCONTROLLED

Type of Furnace	Emission Factor (lb/ton of steel)	Chemical Composition
Open Hearth		
Not Oxygen-Lanced	8	Fe_2O_3 SiO_2 Al_2O_3 CaO MnO P_2O_5 S Fluorides
Oxygen-Lanced	21	Fe_2O_3 FeO SiO_2 Al_2O_3 CaO MnO P_2O_5 MgO S
Basic Oxygen	40	Fe_2O_3 FeO Mn SiO_2 Al_2O_3 CaO MgO P_2O_5 C S
Electric Arc	10	Fe_2O_3 FeO Cr_2O_3 SiO_2 Al_2O_3 CaO MgO MnO ZnO CuO NiO PbO C S P

e. *Basic Oxygen*

The basic oxygen furnace (BOF) is pear shaped, lined with refractory material, and open at the top. The entire furnace tilts for charging and discharging. The furnace is first charged with scrap steel, immediately followed by molten iron (65% to 80% of the charge is molten iron). After being charged and when the furnace is in a vertical position, an exhaust hood is lowered to cover the top and an oxygen lance is lowered through the hood into the furnace to about 6 feet above the charge. Oxygen is injected through the lance at flows up to 6,000 cfm and at pressures up to 160 psi. Later, fluxes are added and the steel is refined at about 5000°F (3000°C). As the impurities are oxidized, they combine with the flux to create the floating slag. After 45 minutes, the content of the steel is tested and the steel is poured through the taphole into a ladle. The taphole holds back the slag, which is later emptied for disposal. As the steel is tapped, alloys are added to the ladle.

The off-gases from the BOF process carry a very heavy particulate load. Figure VI-2 and Table VI-10 describe the concentration and size distribution of the particulates. Gaseous emissions include about 0.2 lb/ton of product (0.1 kg/MT) of fluorides. All BOF processes have particulate emission controls; they are electrostatic precipitators, venturi scrubbers, and baghouses. Excess air is often used to cool the gases before they reach the controls.

f. *Electric Arc*

An electric-arc furnace is a refractory-lined cylinder, capable of handling from 20 to 400 tons. On the larger, newer furnaces the roofs are removable for charging, and doors in the front are for small additions and charging the smaller furnaces. Three carbon electrodes are inserted through the roof to just above the charge, and an arcing electric current supplies the heat. Sometimes oxygen is lanced into the furnace. Tapholes for discharging the steel are either in the bottom for gravity draining or above the charge requiring the furnace to be tilted.

Once the roof of the furnace is swung out of the way, it is charged with scrap and pig iron. The roof is then replaced and the electrodes are lowered. Electricity arcs between metal and the electrodes, creating an intense heat. After the charge melts, the flux is added and an oxidizing slag is formed. During this process, oxygen may be lanced across the furnace. High-quality steel requires two slag-forming periods. Consequently, the initial slag is tapped off and more flux is quickly added to form a second slag under a slightly positive pressure. The steel is not oxidized further in the second slag period. When the analysis of the steel is correct, the furnace is tipped and the steel poured out.

The emission gases include oxygen, carbon dioxide, carbon monoxide, nitrogen and fluorides. Particulate emissions are described in Figure VI-2 and Table VI-10. Baghouses are the control device of choice for the small particles generated by the process.

3. *Prior Evidence of Disease Associated with Industry*

Several investigators have linked the steel industry to various health effects, including neoplasms. These studies have considered both occupational and environmental exposure. Several studies will be discussed here, while a more encompassing review may be found in Appendix H.

Cecilioni has performed several studies relating cancer mortalities to the presence of the steel industry in Hamilton, Ontario [1,2]. The cancers studied were respiratory, gastrointestinal, and genitourinary. He found that:

> "A review of the mortality rates for cancer for the years 1966 to 1970 in Hamilton reveals a considerably higher death rate from cancer in Hamilton than in the less industrialized city of Ottawa. The highest rate (65 per 100,000) occurred in the proximity of the steel mills, compared with the death rates (23 and 12 per 100,000) farther distant." ([2], p. 153)

Since the steel industry produces fluoride contamination and several studies have implicated fluoride in cancer, Cecilioni analyzed the area for fluoride. He found fluoride in vegetation, especially in the steel mill area. In addition, skeletal fluorosis in cancer patients was 3 to 5 times the normal level.

Although Cecilioni's work does not unequivocally prove that the fluoride from steel mills causes cancer in the adjacent community, his findings are very strong and fulfill several important criteria in establishing causality in suspect industry-disease relationships. These criteria are: (1) dose-response (higher mortality rates around steel mills); (2) identification of a specific industrial agent (fluoride); (3) measurement of excessive levels of the emission in the environment; (4) elucidation of a potential pathway to the affected population; and (5) impact on the affected population (e.g., skeletal fluorosis).

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[1] Cecilioni, V. A., Lung cancer in a steel city, its possible relation to fluoride emissions, *Fluoride* 5(4):172-187, 1972.

[2] Cecilioni, V. A., Further observations on cancer in a steel city, *Fluoride* 7(3):153-156, 1974.

Lloyd et al. studied 50,072 steelworkers in seven U.S. plants and analyzed the relationship between work in specific areas of industry and death from all causes during the period 1953 to 1961 [3-8]. The areas were studied for observed and expected deaths from malignant neoplasms, vascular CNS lesions, and heart diseases. The authors conclude:

> "Two exceptions which did *not* appear to be due to selection for health were the excess mortality from vascular lesions of the central nervous system for men employed in sheet finishing and shipping and excess mortality from malignant neoplasms of the respiratory system for nonwhite coke plant workers. It is suggested that these differences may be due to factors in the working environment."([6], p. 157)

Using the steelworker mortality data of Lloyd et al., Mazumdar et al. calculated an index of cumulative exposure for coke oven workers [9]. They concluded that the excess of respiratory cancer observed in this group is related, as expected, to both level of exposure and length of time exposed. They conclude that:

> "Even though the specific carcinogenic element or elements in the coke oven environment are not known at the present time, increasing levels of exposure to coal tar pitch volatiles are related to an increased risk of dying from malignant neoplasms. Further coal tar pitch volatile measurements need to be made and analyses designed which will specify the dose-response relationship more precisely."

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[3] Lloyd, J. W., and A. Ciocco, Long-term mortality study of steelworkers. I. Methodology, *J. Occup. Med. 11*(6):299-310, 1969.

[4] Robinson, H., Long-term mortality study of steelworkers. II. Mortality by level of income in whites and non-whites, *J. Occup. Med. 11*(8):411-416, 1969.

[5] Redmond, C. K., E. M. Smith, J. W. Lloyd, and H. W. Rush, Long-term mortality study of steelworkers. III. Follow-up, *J. Occup. Med. 11* (10):513-521, 1969.

[6] Lloyd, J. W., F. E. Lundin, Jr., C. K. Redmond, and P. B. Geiser, Long-term mortality study of steelworkers. IV. Mortality by work area, *J. Occup. Med. 12*(5):151-157, 1970.

[7] Lloyd, J. W., Long-term mortality study of steelworkers. V. Respiratory cancer in coke plant workers, *J. Occup. Med. 13*(2):53-68, 1971.

[8] Redmond, C. K., A. Ciocco, J. W. Lloyd, and H. W. Rush, Long-term mortality study of steelworkers. VI. Mortality from malignant neoplasms among coke oven workers, *J. Occup. Med. 14*(8):621-629, 1972.

[9] Mazumdar, S., C. Redmond, W. Sollecito, and N. Sussman, An epidemiological study of exposure to coal tar pitch volatiles among coke oven workers, *J. Air Pollut. Control Assoc. 25*(4):382-389, 1975.

In the largest iron and steel works in Japan, six cases of lung cancer were found among coke oven workers from 1947 to 1971. An epidemiological survey [10] indicated that the lung cancer incidence was higher among retired blast furnace and coke oven workers in the iron and steel works than in the general Japanese male population. The ratio of lung cancer cases to malignant neoplasia of all sites was also greater for blast furnace and coke oven workers.

Benzo[a]pyrene, a known carcinogenic agent, was measured by Tanimura [11] and found in quantities from 10- to 60-fold higher at steel production sites than in the air of a neighboring industrial town. These findings document the occurrence of environmental contamination by carcinogenic agents involved in iron and steel production.

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[10] Sakabe, H., K. Tsuchiya, and N. Takekura, Lung cancer among coke oven workers. A report to Labor Standard Bureau, Ministry of Labor, Japan, *Ind. Health 13*(1-2):57-68, 1975.

[11] Tanimura, H., Benzo[a]pyrene concentrations at Japanese iron and steel works, *Arch. Environ. Health 17*(2):172-177, 1968.

C. STUDY DESCRIPTION (ACTIVITIES AND METHODS)

1. General Methodology (Hypothesis Development and Test)

As mentioned previously, a two-stage methodology was used for this study. The first stage consisted of developing hypotheses linking the steel industry with elevated community mortality at Johnstown, Pennsylvania, a site chosen specifically for this purpose. The second stage involved studying Provo, Utah, a site chosen primarily to test the hypotheses previously developed.

The general criteria used in the selection of the hypothesis development site are as follows:

- high industrial activity for the steel industry,
- lack of industrial clutter,
- existence of sufficiently large population at risk, and
- significantly higher mortality rates for digestive neoplasms.

The first three criteria above are those used in the selection of the hypothesis test site. Table VI-11 shows how Johnstown and Provo meet these criteria.

Table VI-11

SITE SELECTION CRITERIA FOR THE STEEL INDUSTRY HYPOTHESIS DEVELOPMENT AND TEST SITES

Criterion	Johnstown	Provo
1. High Steel Activity	Industrial index: 3082 Employs: 14,000	Industrial index: 634 Employs: 5,000
2. Lack of Industrial Clutter	Some clutter: small firms Coal outside city in Cambria County	Only a few small firms
3. Sufficient Population	42,000	53,000
4. Elevated Mortalities (Digestive Neoplasms)	Age-adjusted mortality rates: WM - 120 WF - 75	Not relevant to test site selection

The following sections describe the methods used to establish the hypotheses in Johnstown and then test them in Provo. A site description and site-specific industry characterization are included for each site as well as data collection and analyses.

2. *Hypothesis Development—Johnstown, Pennsylvania*

a. *Site Description*

Johnstown, Pennsylvania, a city of 42,000 people, is located in a narrow Y-shaped valley formed by the confluence of the Stonycreek and Little Conemaugh Rivers in Cambria County. The downtown business section of the community lies in this valley at the point of the confluence and the residential areas have grown up along the branches of the rivers and on the plateaus above the town. The city is 70 miles east of Pittsburgh, 38 miles west of Altoona, and 75 miles north of Cumberland, Maryland. Johnstown's average annual precipitation is 51 inches. The normal temperature range is from 10° to 60°F in the winter to 50° to 90°F in the summer. The elevation in the valley is about 1,200 feet, while elevations on the ridges are around 2,000 feet. Figure VI-3 shows the layout of the Johnstown Air Basin Region.

Johnstown, originally the site of an Indian village, was founded in 1800 by Joseph Johns. In 1834 it was made a borough and was incorporated as a city in 1889. Johnstown is most noted for its floods. First inundated in 1808, Johnstown has since been flooded 18 times. In 1889 the Great Flood occurred when the South Fork Dam breached completely and 500 million cubic feet of water inundated the valley, causing the loss of 2,200 lives and $10 million worth of property. In 1936 the rivers rose and caused a flood that, while killing only 40 people, brought about $40 million worth of property damage. Most recently, in July of 1977, another flood caused by heavy rains inundated the valley. The damage from the most recent flood included the closing of the steel plants, perhaps permanently.

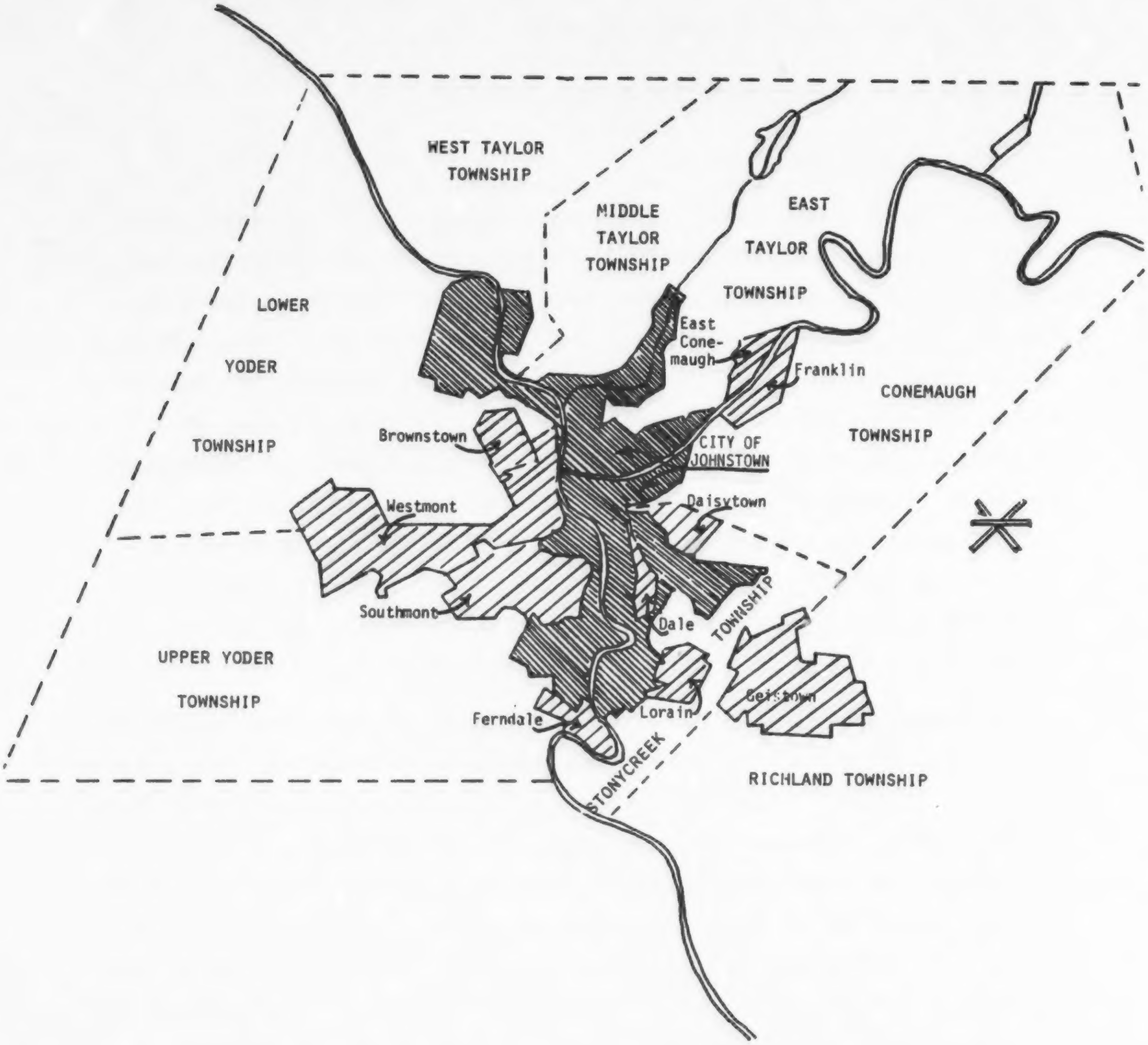

Figure VI-3. Communities in the Johnstown Air Basin

b. Site-Specific Industry Characterization

The steel industry has a long history in Johnstown due to its deposits of iron ore, coal, and limestone. In 1976, about 14,000 people were employed in the steel industry, 12,000 at Bethlehem and 2,000 at U.S. Steel. Figure VI-4 shows the location of the steel industry in the air basin.

The first forge was built in Johnstown in 1809; the first large-scale ironworks in 1840. Steel was first made by the Cambria Iron Co. in 1852. The plant changed hands throughout the early years, finally being purchased by Bethlehem Steel Co. in 1923. The main product of the plant in the early years was steel rails. The first by-product works, designed primarily to make coke for blast furnaces, was erected in 1895 at Johnstown. The Franklin Works, the largest plant in Johnstown, was built around 1898.

Table VI-12 describes the production capacity history of the Bethlehem and U.S. Steel plants in Johnstown.

A steel facilities inventory of the Bethlehem and U.S. Steel plants in the Johnstown area before July 1977 is found in Table VI-13. Emission controls for these furnaces and the sinter plants came on line in the 1960's and 1970's. Although control efficiences indicated in Table VI-13 were high, overall emissions were excessive as shown in Table VI-14.

The Bethlehem plant in Johnstown discharges alarmingly large amounts of toxic heavy metals, especially in its water outfalls, where about 26 lb/day of cadmium, 39 lb/day of arsenic, and 912 lb/day of lead are discharged. Although the population of Johnstown is not exposed via a water route, the Bethlehem Johnstown Works has exceeded Council on Economic Priorities criteria for discharges of 18 out of 20 pollutants, including all the potentially poisonous heavy metals and cyanide. U.S. Steel's Johnstown Works, a much smaller plant, is not a major polluter; Bethlehem's Johnstown Works emits 82 times as much soot and dust as the U.S. Steel plant.

An analysis of industrial polluting sources, including the steel works, done by Barry Evans of EPIC for the Johnstown area may be found in Appendix I. This study relates the presence and changes in emission sources from 1939 to 1977.

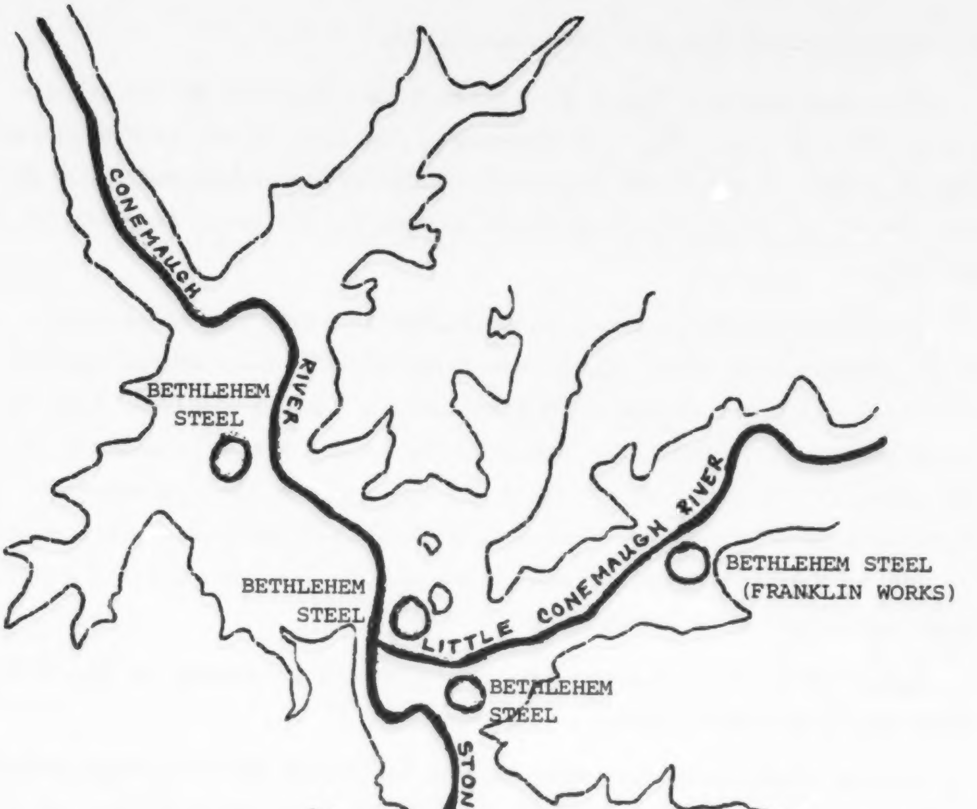

Figure VI-4. Location of Steel Industry in Johnstown Air Basin

Table VI-12. *JOHNSTOWN STEEL INDUSTRY—PRODUCTION CAPACITY HISTORY*

Year	U.S. STEEL			BETHLEHEM STEEL										
	STEEL FURNACES			COKE PLANT			BLAST FURNACES		STEEL FURNACES			ROLLING & PLATE MILLS		
	Furnaces		Annual Capac.[a]	Ovens		Annual Capac.[a]	No.	Annual Capac.[a]	Furnaces		Annual Capac.[a]	Machines		Annual Capac.[a]
	Type	No.		Type	No.				Type	No.		Type	No.	
1901	N.D.	N.D.	N.D.	BP	160	N.D.	6	650,000	BS	N.D.	600,000	N.D.	N.D.	300,000[b]
				BH	920				OH	N.D.	225,000	N.D.	N.D.	324,000[c]
1952	OH	2	18,900	BP	436	2,268,000	N.D.	1,674,000	OH	24	2,028,000	R	15	4,573,000
	EA	2	5,500									PL	2	390,000
1956	OH	2	19,000	BP	436	2,268,000	N.D.	1,664,000	OH	24	2,280,000	R	16	5,740,000
	EA	2	6,000									PL	2	400,000
1958	OH	2	19,000	BP	316	1,836,000	7	2,046,000	OH	21	2,400,00	N.D.	N.D.	N.D.
	EA	2	6,000											
1962	OH	2	19,000	BP	316	1,836,000	6	2,000,000	N.D.	N.D.	2,400,000	R	N.D.	5,385,000
	EA	2	6,000									PL	N.D.	490,000
1963	N.D.	N.D.	N.D.	BP	316	N.D.	6	2,000,000	OH	20	2,400,000	N.D.	N.D.	N.D.
1965	OH	2	N.D.	BP	316	N.D.	6	N.D.	OH	15	N.D.	R	14	4,955,000
	EA	2	N.D.									PL	2	490,000
1970	EA	3	N.D.	BP	316	N.D.	5	N.D.	OH	8	N.D.	R	14	5,664,500
												PL	2	500,000

[a] Annual Capacity in net tons.

[b] Steel rails.

[c] Structural steel.

N.D. = No Data
BP = By-Product
BS = Bessemer
BH = Beehive
OH = Open Hearth
R = Rolling
EA = Electric Arc
PL = Plate

Table VI-13. STEEL FACILITIES INVENTORY FOR JOHNSTOWN, PENNSYLVANIA

FACILITY		BETHLEHEM STEEL JOHNSTOWN WORKS	U.S. STEEL JOHNSTOWN WORKS
COKE OVENS	Number	316	0
	Year Installed	1922	-
BLAST FURNACES	Number	5	0
	Year Installed	Prior to 1900	-
STEEL FURNACES	Number/Type/Size	8/Open Hearth, O_2-Lanced/180 tons each	1/Electric Arc/30 tons 2/Electric Arc/3 tons each
	Year Installed		1966 (30 ton) 1924 (3 ton) 1922 (3 ton)
SINTER PLANT	Number	2	0
	Year Installed		-
CONTROLS	Year Installed Location & Type (% Efficiency)	1973 Blast Furnace - Baghouse 1968 Sinter Plant - ESP (98%) 1972 Sinter Plant - Scrubber 1964 Open Hearth - ESP (98.2%) 1969 Open Hearth - ESP (98.2%)	1966 30-ton Electric Arc - Baghouse (95.0%) 1968 Both 3-ton Electric Arc - to above Baghouse

Table VI-14. STEEL INDUSTRY: AIR EMISSION INVENTORY (tons/year)

Emission	Johnstown, Pennsylvania	
	Bethlehem Steel	U.S. Steel
Particulates	8,600	100
SO_2	13,700	0
NO_x	190	120

c. Data Collection and Analysis

As seen in Table VI-15, the population of the City of Johnstown has been declining over the years, while the population of the metropolitan area has increased. The decrease in population is due, in part, to the lack of industrial diversity which causes young people to move elsewhere to seek employment. This has resulted in an older population in Johnstown. Tables VI-16 and VI-17, obtained from 1970, file C, fifth count census tapes, show the age/race/sex distribution of the population in Johnstown and some summary demographic statistics for Cambria County.

Table VI-15

TOTAL POPULATION OF JOHNSTOWN, PENNSYLVANIA AND ITS METROPOLITAN AREA

POPULATION		
Year	Johnstown	Metropolitan Area
1920	67,327	
1930	66,993	105,000
1940	66,668	
1950	63,232	
1960	53,949	112,641
1970	42,476	262,822

Table VI-16

AGE/RACE/SEX DISTRIBUTION OF 1970 POPULATION OF JOHNSTOWN, PENNSYLVANIA

POPULATION DISTRIBUTION BY AGE, RACE AND SEX				
Age Group	White Male	White Female	Nonwhite Male	Nonwhite Female
0-2	787	762	97	75
3-14	3,957	3,852	459	331
15-24	2,727	3,384	253	281
25-34	1,615	1,864	92	157
35-44	1,885	2,213	95	138
45-54	2,698	3,211	103	166
55-64	2,204	2,863	158	133
65-74	1,340	2,245	51	45
75 and over	877	1,259	55	44
Total	18,090	21,653	1,363	1,370
All ages, WM + WF + NWM + NWF = 42,476				

The areas used for hypothesis development, those labeled Exposed and Unexposed on Table VI-17, were Johnstown, Dale, East Conemaugh, and Franklin compared to Cambria County outside the air basin. The demographic indices for these areas matched within 10%, except for median age. The difference in ages should not be a confounding factor in our analysis, since we are using age-adjusted mortality rates for our comparison.

Development of a plausible hypothesis for health effects requires a plausible pathway for transport of atmospheric pollutants from the plant to the population at risk. This was essentially a meteorological problem, for which two approaches will be discussed. The first approach relied on investigations published in the scientific literature. The second approach made use of available modeling methodologies to calculate a theoretical average concentration of various pollutants at various distances from the factory.

Table VI-17. *SUMMARY DEMOGRAPHIC STATISTICS, 1970 CENSUS, FOR CAMBRIA COUNTY, PENNSYLVANIA*

AREA	Median Age	Median Family Income	% In Same House as in 1965	% White	% With Less Than 1 Person per Room	% Completing High School	% Female	% Native-Born of Native Parents
Johnstown	39.5	8,500	67.1	93.6	94.7	40.1	54.2	74.9
Brownstown	29.5	8,500	79.9	99.4	89.5	44.8	52.9	66.0
Daisytown	29.5	8,500	76.7	99.8	91.1	33.5	47.1	74.7
Dale	29.5	8,500	66.2	98.9	97.4	48.4	54.4	87.1
E. Conemaugh/Franklin	39.5	8,500	73.3	89.9	91.6	38.2	52.2	70.6
East Taylor Township	29.5	8,500	69.6	97.8	94.1	46.5	50.5	80.7
Ferndale	39.5	9,500	70.2	100.0	97.7	60.4	54.4	85.7
Geistown	29.5	11,000	70.3	100.0	96.8	59.0	51.6	81.3
Lorain/Stony Creek Township	29.5	9,500	65.5	99.9	94.3	53.3	51.5	85.1
Lower Yoder Township	29.5	8,500	84.2	96.9	86.3	36.9	50.6	67.9
Middle Taylor Township	29.5	8,500	81.4	96.9	87.9	34.2	50.4	75.6
Southmont	39.5	11,000	75.6	99.4	95.7	74.6	53.0	81.2
Upper Yoder Township	29.5	11,000	61.7	97.8	96.6	69.5	51.6	89.0
Westmont	39.5	13,500	68.0	100.0	98.9	75.8	53.3	75.4
Exposed[a]	39.5	8,500	67.5	93.5	94.7	40.4	54.1	75.2
Unexposed[b]	29.5	8,500	71.4	99.6	91.9	42.1	50.8	80.7

[a] Exposed = Johnstown, Dale, E. Conemaugh/Franklin

[b] Unexposed = Cambria County, except Johnstown Air Basin (Johnstown plus boroughs and townships above = Air Basin)

The two reports in the scientific literature [12,13] were parts of an integrated study performed by Pennsylvania State University's Department of Meteorology. The study attempted to correlate meteorological variables with urban air pollution data in Johnstown. Air quality data on particulates and meteorological parameters (wind speed and direction) were collected during the initial phase of the study.

In the first study reported by Panofsky and Prasad, a diurnal variation in both vertical mixing potential (measured by fluctuation of the bivane) and pollution (as measured by soiling tape transparencies) were reported for clear skies, with greatest particulate concentrations occurring at night. This pollution was attributed primarily to low-level sources. Concerning the correlation of pollution from the Franklin Works with meteorological data, the authors note that,

> "...it is likely that, on a clear day with east or northeast winds, fumigation of particulates from the Franklin Works may become important. However, easterly winds are usually accompanied by thick clouds, often with precipitation." [12]

A mathematical model invoked by Panofsky and Prasad demonstrates, however,

> "...that the huge Franklin Works do significantly affect the air pollution at Johnstown, but only in the case of the relatively infrequent east winds. Of course, the low-cost residential areas to the northeast and east of the Franklin Works encounter serious air pollution problems." [12]

The second investigation was carried out by a student at Penn State University who performed a meteorological experiment using a zinc cadmium sulfide tracer to chart the wind flow in the Stony Creek and Conemaugh River Valley [13]. The purpose of the tracer study was to attempt to correlate meteorological variables with urban air pollution.

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[12] Panofsky, H. A., and B. Prasad, The effect of meteorological factors on air pollution in a narrow valley, *J. Appl. Meteorol.* 6:493-499, 1967.

[13] Smith, D. B., Tracer study in an urban valley, *J. Air Pollut. Control Assoc. 18*(9):600-604, 1968.

In a previous study [14] it was concluded that air pollution was greatest at night when the sky was clear, winds were calm, and vertical ventilation was weakest. Therefore, the subsequent tracer study was performed on calm, clear autumn nights. As the map in this chapter shows (see Figure VI-4), Johnstown is located at the confluence of the Little Conemaugh River and Stony Creek where they merge to form the Conemaugh River. The predominant nocturnal winds under the stated conditions tend to follow typical nocturnal drainage flow from Stony Creek to the north over Johnstown. Thus, the point of release for the tracer was in the middle of Stony Creek, south of the confluence of the rivers. Figure VI-5 illustrates the approximate tracer concentration isopleths as reported by Smith [13]. As can be noted in the abrupt stoppage of tracer flow toward the northeast, the Franklin Works forms an effective heat island which becomes a barrier to tracer flow as material is carried aloft by thermal effluent from the steel plant.

The implications of these two studies, from the standpoint of the present epidemiologic investigation, are twofold. First, the effect of pollution from the Franklin Works should be most severely felt by residents of the Boroughs of East Conemaugh and Franklin, located east and northeast of the plant. In addition, emissions from the Franklin Works would be expected to significantly affect air quality in the City of Johnstown to the west and south.

The air quality data available for Johnstown were quite complete (see Table VI-18). The Pennsylvania Department of Environmental Resources had air quality reports for trace metals as well as criteria pollutants from six sampling stations throughout the Johnstown Air Basin. In addition, a special study they had done of the area [15] produced data on benzo[a]pyrene (BaP) and benzene-soluble organics (BSO) throughout the air basin.

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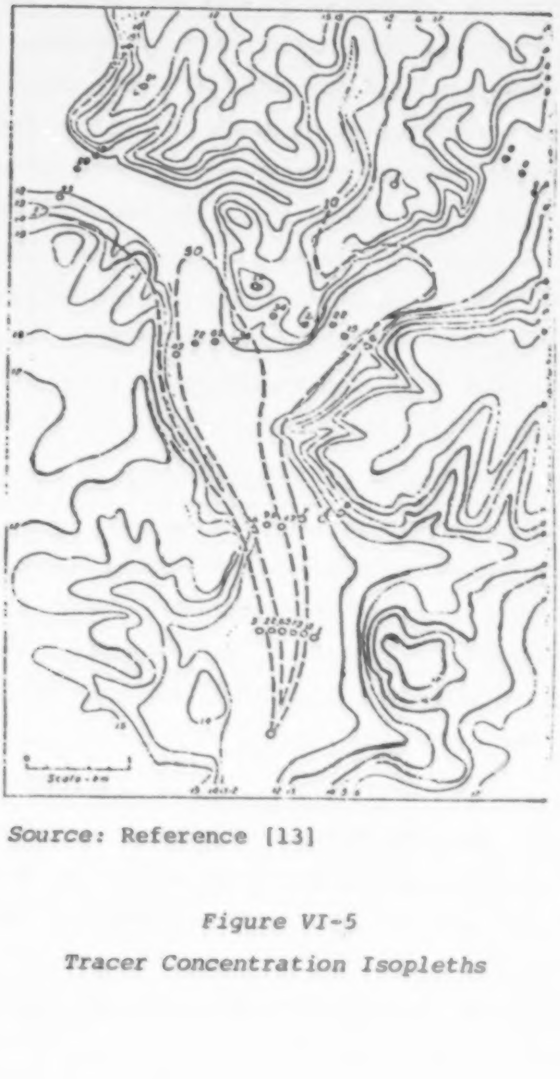
[14] Pennsylvania State University, Department of Meteorology, *Meteorological Effects on Air Pollution at Johnstown, Pa.*, Interim Report 1964-1965.

[15] Pennsylvania Department of Environmental Resources, *Johnstown Air Basin Air Quality Study, Johnstown, Pa.*



Source: Reference [13]

Figure VI-5

Tracer Concentration Isopleths

Table VI-18

AIR QUALITY DATA FOR THE JOHNSTOWN, PENNSYLVANIA, AIR BASIN (ARITHMETIC MEANS & STANDARD DEVIATIONS)

PARAMETER (unit):years data collected	Sampling Station Locations					
	Westmont Borough	Johnstown, South	Dale Borough	Johnstown, North	Johnstown, Central	East Conemaugh Borough
TSP ($\mu g/m^3$):1972-1976	42 ± 3.5	69 ± 6.0	94 ± 9.2	80 ± 21.7	104 ± 8.1	158 ± 8.0
Dustfall ($ton/mi^2/mo$):1972-1976	12 ± 1.3	17 ± .9	26 ± 3.1	19 ± 1.2	26 ± 2.6	65 ± 6.5
Sulfate ($\mu g/m^3$):1972-1976	11.7 ± 1.94	14.0 ± 2.51	13.9 ± 2.89	16.1 ± 2.70	15.9 ± 3.08	19.9 ± 2.65
Nitrate ($\mu g/m^3$):1972-1975	1.67 ± .84	2.25 ± .80	2.59 ± 1.11	2.59 ± 1.25	2.88 ± 1.24	3.24 ± .72
Fluoride ($\mu g/m^3$):1972,1975	.07 ± .08	.11 ± .10	.10 ± .09	.08 ± .08	.09 ± .08	.27 ± .16
Chloride ($\mu g/m^3$):1973	.33	1.24	2.09	1.54	1.83	2.32
BSO ($\mu g/m^3$):1975	2.2	3.9	4.1	5.5	5.4	9.7
BaP (ng/m^3):1975-1976	2.3 ± 1.84	5.4 ± .86	5.0 ± 2.57	15.5 ± 2.36	6.1 ± 2.33	19.9 ± 5.02
Lead ($\mu g/m^3$):1972-1975	.193 ± .024	.629 ± .057	1.02 ± .184	.683 ± .086	.970 ± .164	1.150 ± .197
Cadmium ($\mu g/m^3$):1974-1975	.003 ± .001	.004 ± .001	.004 ± .001	.005 ± .001	.007 ± 0	.005 ± .001
Nickel ($\mu g/m^3$):1972,1975	.009 ± .002	.011 ± .004	.010 ± .006	.010 ± .004	.018 ± .006	.018 ± .001
Iron ($\mu g/m^3$):1972,1975	.785 ± .333	1.328 ± .116	1.749 ± .012	2.679 ± .733	2.580 ± .410	9.479 ± 3.31
Manganese ($\mu g/m^3$):1972,1975	.073 ± .047	.147 ± .009	.257 ± .105	.716 ± .186	.685 ± .105	.924 ± .158

The basic trend for all pollutants seemed to be that the areas high on the valley walls and ridges, as exemplified by Westmont, were not exposed to high emission levels; while the areas closest to the Franklin Works, Franklin and East Conemaugh, were most highly exposed. The rest of the valley seemed to have uniformly distributed pollutant levels. This is consistent with a transport analysis done for this study by Barry Evans of EPIC. A more detailed description of this analysis plus concentration estimates may be found in Appendix I.

Water quality data were not considered for this analysis, not because there was no pollution caused by the steel industry, but because the water supply for the Johnstown area and the downstream communities comes from sources unaffected by steel industry effluents.

Mortality data from 1965 through 1975 were used for the Johnstown analyses. They were obtained on tape from the Pennsylvania Department of Health.* The following items were coded on the tape:

State file number
Place where death occurred
Residence of decedent
Hospital where death occurred
Cause of death
Place of accident (if accidental death)
Date of death
Sex of decedent
Race of decedent
Date of birth of decedent
Age of decedent
If an autopsy was performed

Conversations with Mr. Ed Digon of the Pennsylvania Department of Health, Division of Epidemiologic Research, revealed a possible residency-reporting problem on death certificates from the Johnstown Air

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*Basic data for use in this study were supplied by the Pennsylvania Department of Health, Harrisburg, Pennsylvania. Any analysis, interpretation, or conclusion based on these data is solely that of Enviro Control, Inc., and the Pennsylvania Department of Health specifically disclaims responsibility for any such analysis, interpretation, or conclusion.

Basin. A study done by them for a single year of data showed an 18% overreporting of Johnstown as the place of residence of the decedent. To correct for this potential problem, we obtained actual street addresses from about 900 hard copy death certificates which were selected by cause of death. After using an R. F. Polk Directory and detailed street maps to verify actual residence, we found the error to be only 9% over the 11 years of data we examined.

We had hoped to obtain morbidity data from the County Tumor Registry from the Conemaugh Valley Memorial Hospital. Unfortunately, before our request had been processed the area was flooded and the Tumor Registry staff was unable to provide the requested information. Due to these unfortunate circumstances, all mortalities from malignant neoplasms were calculated from death certificate data. No incidence or severity data were available.

Average annual age-specific mortality rates for all age groups and average annual age-adjusted mortality rates for ages 35-74 were calculated per 100,000 population, as described in Appendix J. The age-adjusted mortality rates for the exposed (Johnstown, Dale, East Conemaugh and Franklin) and unexposed (Cambria County except the Johnstown Air Basin) areas were compared using the Mantel-Haenszel test [16]. The results of these calculations can be seen in Table VI-19.

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[16] Mantel, N., and W. Haenszel, Statistical aspects of the analysis of data from retrospective studies of disease, *J. Nat. Cancer Inst.* *22*(4):719-748, April 1959.

Table VI-19. *COMPARISON OF AGE-ADJUSTED MORTALITY RATES FOR JOHNSTOWN, PENNSYLVANIA (using the Mantel-Haenszel χ^2 test)*

CAUSE OF DEATH (ECI #)	White Males				White Females			
	Mortality Rate		Relative Risk	Level of Signif.	Mortality Rate		Relative Risk	Level of Signif.
	Exposed	Unexposed			Exposed	Unexposed		
Infectious & parasitic diseases(1)	29	23	1.19	NS	10	8	1.34	NS
Cancer, oral, pharynx & esophagus(2)	33	20	1.74	.025	6	0	11.93	.01
Cancer, stomach(3)	40	26	1.60	.05	11	7	1.55	NS
Cancer, duodenum & small intestine(4)	0	0	--	--	1	0	--	--
Cancer, colon & rectum(5)	64	49	1.31	NS	42	34	1.20	NS
Cancer, liver & bile ducts(6)	10	8	1.31	NS	14	9	1.42	NS
Cancer, pancreas(7)	32	20	1.59	NS	9	9	--	--
Other digestive cancers(8)	0	0	--	--	6	1	4.26	.05
Cancer, respiratory organs(9)	199	94	2.13	.005	26	12	2.22	.005
Cancer, genitourinary organs except bladder(10)	40	30	1.40	NS	44	49	--	--
Cancer of the bladder(11)	17	14	1.23	NS	3	2	1.18	NS
Other cancers except leukemia(12)	73	48	1.49	.025	111	85	1.30	.025
Neoplasms of lymphatics(13)	52	30	1.65	.025	25	23	1.17	NS
Endocrine, nutritional, metabolic & blood disease(14)	78	44	1.82	.005	61	76	--	--
Mental & nervous system diseases(15)	19	13	1.43	NS	10	12	--	--
Rheumatic heart disease(16)	32	20	1.59	NS	36	27	1.30	NS
Hypertensive disease(17)	36	13	2.68	.005	27	19	1.38	NS
Cardiovascular diseases(18)	1,501	946	1.87	.005	645	482	1.40	.005
Cerebrovascular diseases(19)	194	116	1.72	.005	127	105	1.21	NS
Respiratory diseases(20)	126	116	1.07	NS	40	23	1.64	.025
Bronchitis, emphysema & asthma(21)	73	45	1.60	.005	12	9	1.31	NS
Pneumoconiosis(22)	36	155	--	--	0	0	--	--
Other digestive diseases(23)	3	2	1.36	NS	0	1	--	--
Diseases of stomach & duodenum(24)	21	15	1.32	NS	5	5	--	--
Appendicitis, hernia & others(25)	22	19	1.14	NS	20	14	1.43	NS
Gastroenteritis, noninfectious(26)	0	1	--	--	1	0	--	--
Diseases of liver, gallbladder & pancreas (27)	88	56	1.58	.005	34	27	1.28	NS
Diseases of urinary system(28)	33	23	1.44	NS	18	7	2.58	.005
Diseases of the genital organs(29)	8	3	2.82	NS	1	2	--	--
Diseases of pregnancy & childbirth(30)	0	0	--	--	0	1	--	--
Diseases of the skin & musculoskeleton & ill-defined diseases(31)	19	12	1.62	NS	12	11	1.10	NS
Congenital anomalies(32)	5	3	1.61	NS	4	4	--	--
Perinatal diseases(33)	0	0	--	--	1	0	--	--
Accidents, poisons & violence (34)	116	120	--	--	38	29	1.34	NS

NS = Not significant.

3. *Hypothesis Test—Provo, Utah*

a. *Site Description*

Provo, Utah, a city of 53,000 people, is located in the Utah Valley on the eastern shore of freshwater Utah Lake in Utah County. Provo is the largest city in the county, but several other smaller cities are scattered along the eastern shore of the lake. The city of Provo is 45 miles south of Salt Lake City. Provo's average annual precipitation is 11 inches. The normal temperature range is from 19° to 38°F in the winter, to 55° to 90°F in the summer. Utah Valley is bordered on the east by the Wasatch Mountains (11,000 feet) and on the west by the Lake Mountains (10,000 feet). The elevation in the valley is about 4,500 feet. Figure VI-6 shows the layout of the Provo area.

Provo was founded in 1849 by Mormon settlers from Salt Lake City. At first agriculture was the primary industry, with the good water sources from the eastern mountains being of tremendous assistance for irrigation. Shortly after its founding, Provo began to start manufacturing various products, mostly connected with agriculture. The Knight Woolen Mill was one of the largest firms at this time. In 1875 Brigham Young Academy (now Brigham Young University) was founded. The university has been and remains today one of the major contributors to Provo's economy.

b. *Site-Specific Industry Characterization*

The steel industry has a long history in the State of Utah. Shortly after the first Mormon pioneers arrived in the valley of the Great Salt Lake in 1847, an exploring expedition discovered significant iron and coal deposits in the area which now surrounds Cedar City, in the southern part of Utah.

Plans were laid immediately for starting an iron industry. A colonizing company left Provo in 1850 and journeyed by ox team to the area near what is now Cedar City, where they built Utah's first blast furnace. In 1852,the first pig iron made west of the Missouri River was produced in this furnace.

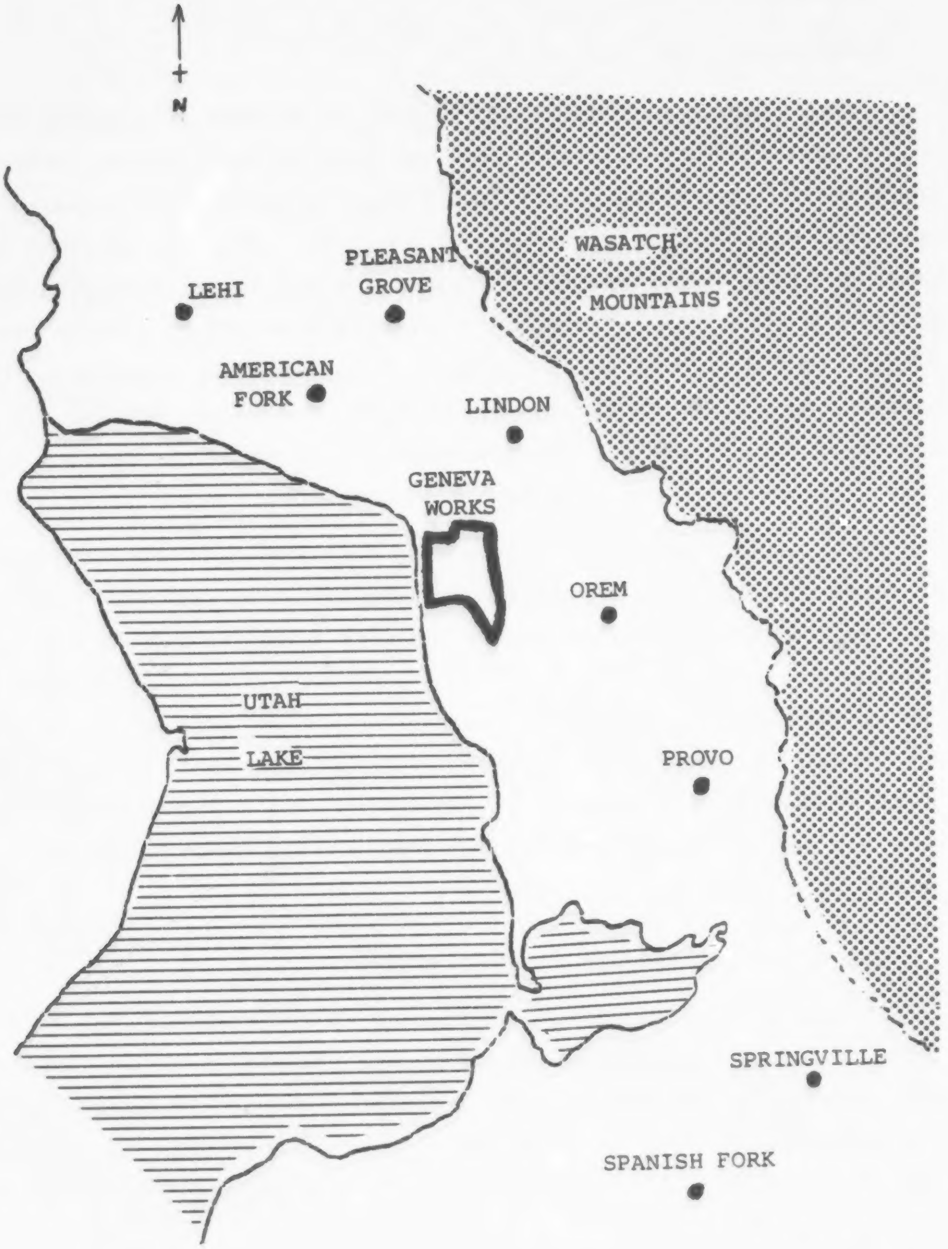

Figure VI-6. Utah Valley

In the meantime, private enterprise organized the Deseret Iron Company to provide capital for development of the iron industry. The Deseret Iron Company purchased the original Utah blast furnace and began to enlarge facilities. But despite their efforts the venture was not a financial success.

Subsequent attempts to start an iron industry in southern Utah were made in 1854 and 1868. The latter continued in production for 15 years, and the pig iron was used to make stoves, grates, iron pots, frying pans, flat irons, buckets, and other castings. But this operation, like those before it, was not a financial success and ceased operation in 1883.

The first financially successful operation to use southern Utah iron ores was begun in 1922 when the Columbia Steel Corporation started construction of a 450-ton blast furnace at Ironton, south of Provo. The first iron was produced at Ironton on April 30, 1924. Pig iron produced at the Ironton furnace was intended primarily for consumption in Columbia Steel Corporation's steel plants at Pittsburg and Torrance in California. The balance of the output was sold to foundries throughout the western states. This arrangement continued following the purchase of the Columbia Steel Corporation by the United States Steel Corporation in 1930.

In 1941-1942, the Federal Government decided to build additional steel production facilities in the West as a precaution against possible closing of the Panama Canal by enemy attacks.

A new steel mill was designed and built without charge or fee by U.S. Steel's Columbia Steel Company. The new mill was constructed in Utah because of adequate transportation; minimum distance from sources of iron ore, coal, limestone, and dolomite; ready access of sufficient fresh water; and safe distance from the vulnerable Pacific Coast. This mill was named the Geneva Plant after the small summer resort of Geneva on the shore of Utah Lake. The first pig iron was smelted at the new plant in January of 1944, followed by the first open-hearth steel a month later.

At the close of World War II, the War Assets Administration offered to sell the Geneva Plant to private industry. U.S. Steel bid for the plant and took over operation of the Utah facilities on June 19, 1946. Since 1946 many facilities have been added to the Geneva Works, nearly doubling its annual steelmaking capacity to 2,500,000 ingot tons per year, and adding a variety of new products such as hot-rolled strip and sheets, welded steel pipe, and synthetic nitrogen chemicals.

Located six miles northwest of Provo, the Geneva Works is one of the larger steelmaking facilities in the west. Geneva's principal products are plates, hot-rolled sheets and coils, structural shapes, welded steel pipe, pig iron, coke, blast furnace and open-hearth slag products, coal chemicals, and nitrogen products for fertilizer and industrial use.

A steel facilities inventory of the Geneva Works is found in Table VI-20. Emission controls for the plant, begun in the 1950's (see Table VI-20), included electrostatic precipitators (ESP) and new coke oven doors and jambs to reduce fugitive emissions. The emissions for the plant are summarized in Table VI-21.

Air pollution control measures were not taken without public pressure, however. In the late fifties, a group of farmers complained to the Utah Department of Agriculture that some of their livestock were afflicted with swollen joints and loss of teeth. An investigation by the State discovered that this condition was due to a type of fluoride poisoning caused by excessive airborne fluoride emissions coming from the blast furnaces and open hearths at the Geneva Works. U.S. Steel eventually agreed to pay damages to the farmers and undertook the building of a wet scrubbing system at the furnaces to supplement the electrostatic precipitators that had been operating since 1956.

Geneva's water pollution control program is quite good; its discharges exceed Council on Economic Priorities (CEP) criteria for only 6 out of 20 pollutants.

Table VI-20. STEEL FACILITIES INVENTORY FOR PROVO, UTAH

FACILITY		U.S. STEEL GENEVA WORKS
COKE OVENS	Number	252
	Year Installed	1944
BLAST FURNACES	Number	3
	Year Installed	1944
STEEL FURNACES	Number/Type/Size	10/Open Hearth/340 tons each
	Year Installed	1944
SINTER PLANTS	Number	2
	Year Installed	1941
CONTROLS		1946 Blast Furnace – Inertial Dust Catcher, Counter Current Scrubber, ESP 1954 Sinter Plant – ESP 1955 Blast Furnace – Orifice Scrubber 1955 Open Hearth – ESP 1962 Sinter Plant – Scrubbers 1962 Open Hearth – Wet Scrubbers 1971 Coke Oven – New Doors, Jambs, etc. 1974 Open Hearth – More Power to ESP 1975 Open Hearth – Collector Modified 1976 Coke Oven – Practices Modified

Table VI-21. STEEL INDUSTRY, PROVO, UTAH: AIR EMISSION INVENTORY (tons/year)

EMISSION	U.S. STEEL, PROVO, UTAH
Particulates	4,700
SO_2	320
NO_x	--

c. *Data Collection and Analysis*

As seen in Table VI-22, the population of Utah County has been increasing steadily over the years. This has been the trend since its founding. Utah, in general, has rapid population growth, and Utah County reflects this trend. Table VI-23 shows the age/race/sex distribution of the population in Provo as determined from 1970, file C, fifth count census tapes. Summary demographic statistics for Utah County communities and the control site, Ogden, which is in Weber County, are shown in Table VI-24.

Table VI-22. UTAH COUNTY: HISTORICAL AND FORECAST POPULATIONS

COMMUNITY	POPULATION					
	1940	1950	1960	1970	1975	1980 Est.*
American Fork	3,333	5,126	6,373	7,713	10,803	12,689
Lehi	2,733	3,627	4,377	4,659	5,597	6,729
Lindon	587	801	1,150	1,644	2,161	2,914
Orem	2,914	8,351	18,394	25,729	35,292	46,767
Pleasant Grove	1,941	3,195	4,772	5,327	6,566	8,127
Provo	18,071	28,937	36,047	53,131	59,729	67,103
Spanish Fork	4,167	5,230	6,742	7,284	8,404	9,539
Springville	4,796	6,475	7,913	8,790	10,764	12,891
Utah County (uninc.)	11,001	11,592	12,056	12,583	14,830	17,568
Other Communities	7,839	8,578	9,437	10,916	13,851	17,900
TOTALS	57,382	81,912	106,991	137,776	167,997	202,227

*Projections based on the 208 Water Quality Study for Mountainlands Association of Governments.

The study area demographics matched those of Ogden within 15% except for three indices in Provo that were outside this limit. These indices—median family income, % in same house as in 1965, and % completing high school—all reflect the presence of Brigham Young University, whose 26,000 enrollment is bound to have an effect on such factors. The use of health data from decedents aged 35 to 74 only should balance this at least in part. The other Utah County communities studied were more closely matched to Ogden demographically.

Table VI-23. AGE/RACE/SEX DISTRIBUTION OF 1970 POPULATION OF PROVO, UTAH

POPULATION DISTRIBUTION BY AGE, RACE AND SEX				
Age Group	White Male	White Female	Nonwhite Male	Nonwhite Female
0-2	1,785	1,562	23	36
3-14	4,136	4,029	40	17
15-24	10,502	12,880	130	226
25-34	3,429	2,570	113	69
35-44	1,450	1,441	9	26
45-54	1,337	1,664	11	0
55-64	1,298	1,427	3	13
65-74	806	998	6	0
75 & over	396	690	0	0
All Ages	25,139	27,261	335	387
All Ages, WM + WF + NWM + NWF = 53,122				

Table VI-24. SUMMARY DEMOGRAPHIC STATISTICS, 1970 CENSUS FOR COMMUNITIES IN UTAH & WEBER COUNTIES, UTAH

TOWN	Median Age	Median Family Income	% In Same House As in 1965	% White	% With Less Than 1 Person per Room	% Completing High School	% Female	% Native-Born of Native Parents
American Fork	23.0	8,500	63.3	100.0	89.5	71.8	51.1	94.5
Lehi	21.0	8,500	62.9	100.0	86.4	70.3	48.9	93.1
Lindon	20.0	9,500	62.7	100.0	76.6	64.5	49.5	95.3
Orem	20.0	9,500	53.0	99.1	86.8	77.6	50.1	91.4
Pleasant Grove	21.0	8,500	70.0	100.0	85.4	64.4	50.4	93.1
Provo	21.0	7,500	30.2	98.6	89.5	78.7	52.0	87.1
Spanish Fork	23.0	8,500	62.7	99.9	90.9	68.3	50.6	92.6
Springville	23.0	8,500	62.4	99.5	89.4	70.5	51.0	92.3
Ogden	23.0	9,500	52.0	95.5	92.6	60.7	51.3	86.2

Meteorology in the Geneva-Provo-Orem area is regulated primarily by the mountains to the north and east and Utah Lake which extends 10 miles to the west. Winds tend to blow out of the mountains and over the lake as is evidenced by the wind rose for Provo presented in Figure VI-7. Data on atmospheric stability were not readily available for Provo or Orem.

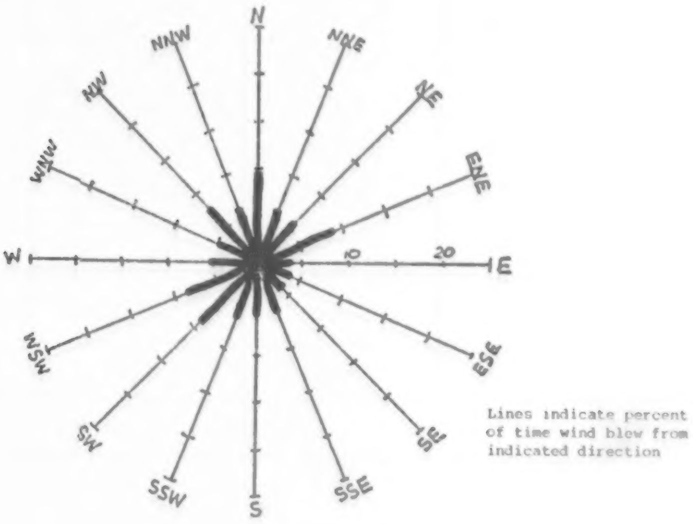

Figure VI-7. Wind Rose for Provo, Utah (1966-1969 data)

A transport analysis using equation (4) in Appendix A was attempted to delineate population at risk and estimate dosage of particulate emissions to which they have been exposed. Since neither STAR data nor stability information was readily available for Orem or Provo, a STAR tabulation from Salt Lake City was used. During plotting of isopleths of calculated long-term concentrations of particulate matter, it became obvious that Salt Lake City's meteorology was inappropriate. The mountains and lake in Salt Lake are oriented differently from those near Orem, such that prevailing winds in Salt Lake blow at right angles to those in Provo-Orem. Thus, the modeling effort was abandoned for this site.

The air quality data for Provo was not as complete as that available for Johnstown. The State of Utah apparently does not have the extensive monitoring programs that Pennsylvania does. We were able, however, through the efforts of Justice Manning at EPA, Research Triangle Park, to obtain BaP data from a special study of the Geneva Works [18]. The data available are presented in Table IV-25.

Table IV-25. AIR QUALITY DATA FOR RELEVANT SITES IN UTAH

SITE	TSP ($\mu g/m^3$)		NO_2 (ppm)
	Ann. Geo. Mean	Max. 24-hr Avg.	
Geneva	73	330	
Lehi	67	302	
Lindon	83	480	
Orem	78	374	
Pleasant Grove	70	531	
Provo	88	481	.028
Ogden	79.3	378	.025

SITE & LOCATION WITH REGARD TO COKE BATTERIES			BaP (ng/m^3)	TSP ($\mu g/m^3$)
1	2.0 km northwest		2.1	124
2	2.7 km northwest		3.8	235
3	2.4 km northwest		3.2	180
4	1.8 km north		2.4	187
5	1.3 km northeast		3.1	136
6	2.4 km southeast		1.6	275
7	4.0 km northwest		2.1	139
8	2.6 km south		1.5	N.D.
9	30.0 km south	Controls	0.1	N.D.
10	20.0 km north		0.8	N.D.

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[18] Stanford Research Institute, *Human Population Exposures to Coke Oven Atmospheric Emissions*, prepared for EPA Contract No. 68-01-4314, by SRI, August 1977.

Utah Valley is a broad valley prone to inversions, particularly in the winter months. While the emissions tend to be more dispersed here than in the narrow Johnstown Valley, there is still a noticeable haze in the valley during the inversions.

Mortality data from 1965 through 1975 were used for the Provo analyses. They were obtained on tape from the Utah Division of Health.* The following items were coded on the tape:

Sex of decedent
Date of death
Race of decedent
Date of birth
Location of death
Marital status of decedent
Residence of decedent
Cause of death
If an autopsy was performed
If a violent death

We were able to obtain, on tape, the cancer registry data for Utah** from 1966 through 1975 from Dr. Joseph L. Lyon of the University of Utah, Department of Family and Community Medicine. We were informed that this tape contained residence information by zip code, which would have enabled us to examine the incidences and mortalities at a more detailed level than the county. Unfortunately, after obtaining the tape, we found that zip code had been coded only for two years of data. There was not enough data for us to analyze successfully. We were thus forced to look at these data on a county level only, comparing Utah County to Weber County; however, we were able to compare incidence and mortality in these two counties.

Average annual age-specific incidence and mortality rates for all age groups and for ages 35-74 were calculated per 100,000 population as described in Appendix J using both the mortality and cancer registry tapes.

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*Basic data used in this study were supplied by the Utah Division of Health, Salt Lake City, Utah. Any analysis, interpretation, or conclusion based on these data is solely that of Enviro Control, Inc.

**These data were supplied by the Utah State Cancer Registry. Any analysis, interpretation, or conclusion based on these data is solely that of Enviro Control, Inc.

The age-adjusted incidence and mortality rates for the test sites were compared to the control areas using the Mantel-Haenszel test as previously described. The results of these calculations can be seen in Tables VI-26 through VI-29.

Table VI-26. *COMPARISON OF AGE-ADJUSTED MORTALITY RATES* FOR WHITE FEMALES IN UTAH (Using the Mantel-Haenszel χ^2 Test)*

CAUSE OF DEATH (ECI #)†	RATE IN OGDEN	AMERICAN FORK			LEHI			LINDON			OREM		
		Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.
1	4	7	2.15	NS	26	7.20	NS	0	--	--	3	--	--
2	3	0	--	--	0	--	--	0	--	--	2	--	--
3	9	13	1.95	NS	13	1.45	NS	0	--	--	5	--	--
4	0	0	--	--	0	--	--	0	--	--	0	--	--
5	22	47	2.19	NS	26	1.16	NS	0	--	--	21	--	--
6	4	0	--	--	12	3.04	NS	0	--	--	0	--	--
7	7	55	7.25	.005	26	3.67	NS	0	--	--	15	--	--
8	0	0	--	--	0	--	--	0	--	--	0	--	--
9	13	21	1.34	NS	12	--	--	0	--	--	0	--	--
10	32	39	1.14	NS	26	--	--	0	--	--	31	--	--
11	2	0	--	--	0	--	--	0	--	--	5	2.54	NS
12	70	101	1.38	NS	105	1.31	NS	0	--	--	81	1.13	NS
13	21	10	--	--	50	2.49	NS	0	--	--	11	--	--
14	41	64	1.64	NS	24	--	--	64	1.22	NS	21	--	--
15	23	11	--	--	0	--	--	0	--	--	20	--	--
16	40	42	0.92	NS	35	--	--	0	--	--	22	--	--
17	10	18	2.00	NS	0	--	--	0	--	--	3	--	--
18	302	404	1.34	NS	281	--	--	110	--	--	314	1.04	NS
19	67	133	2.08	.025	104	1.56	NS	46	--	--	84	1.28	NS
20	30	32	0.99	NS	30	--	--	64	1.66	NS	9	--	--
21	12	11	--	--	0	--	--	0	--	--	9	--	--
22	0	0	--	--	0	--	--	0	--	--	0	--	--
23	2	0	--	--	0	--	--	0	--	--	0	--	--
24	5	0	--	--	0	--	--	0	--	--	5	--	--
25	15	11	--	--	0	--	--	0	--	--	17	1.00	NS
26	0	0	--	--	0	--	--	0	--	--	0	--	--
27	34	42	1.04	NS	0	--	--	0	--	--	25	--	--
28	16	11	--	--	0	--	--	0	--	--	13	--	--
29	1	0	--	--	0	--	--	0	--	--	0	--	--
30	1	0	--	--	0	--	--	0	--	--	0	--	--
31	23	116	4.48	.005	77	3.57	0.01	0	--	--	19	--	--
32	1	0	--	--	0	--	--	0	--	--	4	4.33	NS
33	0	0	--	--	0	--	--	0	--	--	0	--	--
34	55	63	1.16	NS	27	--	--	0	--	--	24	--	--

(continued)

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*Data for 1965 through 1975 from mortality tape.

†See Table VI-19 for definitions of causes of death by ECI #.

Table VI-26 (cont.). *COMPARISON OF AGE-ADJUSTED MORTALITY RATES* FOR WHITE FEMALES IN UTAH*

CAUSE OF DEATH (ECI #)†	RATE IN OGDEN	PLEASANT GROVE			PROVO			SPANISH FORK			SPRINGVILLE		
		Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.
1	4	24	6.10	NS	1	--	--	7	2.03	NS	0	--	--
2	3	0	--	--	3	--	--	0	--	--	0	--	--
3	9	0	--	--	8	--	--	0	--	--	6	--	--
4	0	0	--	--	0	--	--	0	--	--	0	--	--
5	22	24	1.10	NS	23	1.04	NS	22	--	--	30	1.38	NS
6	4	0	--	--	8	1.95	NS	15	3.53	NS	6	1.47	NS
7	7	0	--	--	1	--	--	7	--	--	18	2.61	NS
8	0	0	--	--	2	--	--	7	--	--	0	--	--
9	13	0	--	--	6	--	--	16	1.13	NS	6	--	--
10	32	20	--	--	27	--	--	14	--	--	95	2.90	.005
11	2	0	--	--	3	1.61	NS	0	--	--	0	--	--
12	70	77	1.17	NS	89	1.29	NS	111	1.61	NS	65	--	--
13	21	49	2.24	NS	16	--	--	50	2.47	NS	6	--	--
14	41	63	1.49	NS	24	--	--	80	2.01	NS	31	--	--
15	23	45	1.95	NS	22	--	--	28	1.32	NS	6	--	--
16	40	24	--	--	30	--	--	58	1.47	NS	92	2.27	.01
17	10	0	--	--	9	--	--	0	--	--	0	--	--
18	302	306	1.02	NS	244	--	--	368	1.26	NS	257	--	--
19	67	74	1.09	NS	62	--	--	124	1.91	.025	71	1.10	NS
20	30	13	--	--	11	--	--	0	--	--	32	1.02	NS
21	12	20	2.03	NS	9	--	--	13	1.24	NS	7	--	--
22	0	0	--	--	0	--	--	0	--	--	0	--	--
23	2	0	--	--	0	--	--	0	--	--	0	--	--
24	5	0	--	--	8	1.64	NS	14	3.19	NS	0	--	--
25	15	26	1.59	NS	9	--	--	9	--	--	31	2.03	NS
26	0	0	--	--	0	--	--	0	--	--	0	--	--
27	34	0	--	--	16	--	--	15	--	--	13	--	--
28	16	13	--	--	14	--	--	7	--	--	6	--	--
29	1	0	--	--	0	--	--	0	--	--	6	7.47	NS
30	1	0	--	--	0	--	--	0	--	--	0	--	--
31	23	70	2.90	.05	14	--	--	35	1.63	NS	19	--	--
32	1	0	--	--	1	--	--	0	--	--	0	--	--
33	0	0	--	--	0	--	--	0	--	--	0	--	--
34	55	68	1.23	NS	42	--	--	63	1.13	NS	51	--	--

*Data for 1965 through 1975 from mortality tape.

†See Table VI-19 for definitions of causes of death by ECI #.

Table VI-27. COMPARISON OF AGE-ADUSTED MORTALITY RATES* FOR WHITE MALES IN UTAH (Using Mantel-Haenszel χ^2 Test)

CAUSE OF DEATH (ECI #)†	RATE IN OGDEN	AMERICAN FORK			LEHI			LINDON			OREM		
		Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.
1	14	47	3.83	.01	0	--	--	0	--	--	13	--	--
2	15	0	--	--	0	--	--	0	--	--	19	1.27	NS
3	15	0	--	--	0	--	--	0	--	--	7	--	--
4	0	0	--	--	0	--	--	0	--	--	0	--	--
5	33	43	1.35	NS	25	--	--	0	--	--	21	--	--
6	3	10	2.68	NS	0	--	--	0	--	--	0	--	--
7	19	15	--	--	0	--	--	0	--	--	32	1.43	NS
8	3	0	--	--	13	5.11	NS	0	--	--	0	--	--
9	75	60	--	--	108	1.44	NS	0	--	--	76	0.98	NS
10	30	35	1.19	NS	16	--	--	0	--	--	31	1.02	NS
11	13	0	--	--	0	--	--	0	--	--	0	--	--
12	31	102	3.21	.005	48	1.40	NS	0	--	--	54	1.77	NS
13	36	18	--	--	16	--	--	0	--	--	37	0.90	NS
14	34	60	1.80	NS	63	1.74	NS	0	--	--	22	--	--
15	56	40	--	--	29	--	--	0	--	--	18	--	--
16	25	7	--	--	0	--	--	0	--	--	19	--	--
17	16	7	--	--	0	--	--	0	--	--	13	--	--
18	778	750	--	--	712	--	--	74	--	--	598	--	--
19	101	88	--	--	70	--	--	74	--	--	99	--	--
20	107	82	--	--	95	--	--	0	--	--	60	--	--
21	67	60	--	--	143	2.10	NS	0	--	--	51	--	--
22	2	0	--	--	0	--	--	0	--	--	4	2.88	NS
23	2	0	--	--	0	--	--	0	--	--	0	--	--
24	23	15	--	--	0	--	--	0	--	--	15	--	--
25	20	30	1.69	NS	9	--	--	0	--	--	18	--	--
26	1	0	--	--	16	16.97	NS	0	--	--	0	--	--
27	64	0	--	--	41	--	--	74	.75	NS	26	--	--
28	21	27	1.23	NS	45	2.09	NS	0	--	--	15	--	--
29	1	0	--	--	0	--	--	0	--	--	0	--	--
30	0	0	--	--	0	--	--	0	--	--	0	--	--
31	94	121	1.25	NS	109	1.10	NS	0	--	--	39	--	--
32	2	0	--	--	22	11.75	.025	0	--	--	7	2.41	NS
33	0	0	--	--	0	--	--	0	--	--	0	--	--
34	159	175	1.09	NS	165	1.06	NS	0	--	--	87	--	--

(continued)

[*] Data for 1965 through 1975 from mortality tape.

[†] See Table VI-19 for definitions of causes of death by ECI #.

Table VI-27 (cont.). *COMPARISON OF AGE-ADJUSTED MORTALITY RATES* FOR WHITE MALES IN UTAH*

CAUSE OF DEATH (ECI #)†	RATE IN OGDEN	PLEASANT GROVE			PROVO			SPANISH FORK			SPRINGVILLE		
		Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.	Rate	Relative Risk	Level of Signif.
1	14	11	--	--	7	--	--	0	--	--	12	--	--
2	15	0	--	--	15	--	--	9	--	--	7	--	--
3	15	17	.95	NS	11	--	--	18	1.13	NS	39	2.56	NS
4	0	0	--	--	2	--	--	0	--	--	0	--	--
5	33	0	--	--	27	--	--	26	--	--	21	--	--
6	3	17	4.10	NS	7	2.26	NS	10	2.52	NS	0	--	--
7	19	0	--	--	11	--	--	19	--	--	6	--	--
8	3	17	4.50	NS	2	--	--	0	--	--	0	--	--
9	75	74	--	--	54	--	--	76	1.01	NS	38	--	--
10	30	0	--	--	38	1.26	NS	29	--	--	40	1.28	NS
11	13	0	--	--	2	--	--	0	--	--	0	--	--
12	31	119	3.97	.005	40	1.28	NS	69	2.16	NS	46	1.43	NS
13	36	40	1.10	NS	35	--	--	55	1.72	NS	26	--	--
14	34	99	2.77	.025	26	--	--	103	2.87	.005	70	2.11	.05
15	56	80	1.44	NS	29	--	--	27	--	--	31	--	--
16	25	11	--	--	20	--	--	36	1.31	NS	55	2.02	NS
17	16	51	2.53	NS	11	--	--	26	1.60	NS	0	--	--
18	778	759	--	--	595	--	--	592	--	--	716	--	--
19	101	128	1.35	NS	80	--	--	79	--	--	52	--	--
20	107	62	--	--	66	--	--	53	--	--	62	--	--
21	67	65	--	--	72	1.07	NS	67	--	--	52	--	--
22	2	11	10.83	NS	0	--	--	0	--	--	0	--	--
23	2	0	--	--	0	--	--	0	--	--	15	9.35	NS
24	23	17	--	--	16	--	--	35	1.43	NS	21	--	--
25	20	28	1.28	NS	27	1.41	NS	7	--	--	7	--	--
26	1	0	--	--	0	--	--	0	--	--	0	--	--
27	64	23	--	--	40	--	--	7	--	--	31	--	--
28	21	11	--	--	10	--	--	27	1.23	NS	26	1.22	NS
29	1	17	22.38	NS	0	--	--	0	--	--	0	--	--
30	0	0	--	--	0	--	--	0	--	--	0	--	--
31	94	76	--	--	30	--	--	38	--	--	55	--	--
32	2	10	4.27	NS	3	1.58	NS	0	--	--	0	--	--
33	0	0	--	--	0	--	--	0	--	--	0	--	--
34	159	79	--	--	131	--	--	191	1.18	NS	112	--	--

*Data for 1965 through 1975 from mortality tape.

†See Table VI-19 for definitions of causes of death by ECI #.

Table VI-28. *AGE-ADJUSTED INCIDENCE AND MORTALITY RATES FOR WHITE FEMALES IN UTAH**

CAUSE OF DEATH Site of Neoplasm	INCIDENCE RATES		Relative Risk	Level of Signif.	MORTALITY RATES		Relative Risk	Level of Signif.
	Utah County	Weber County			Utah County	Weber County		
Oral, pharynx, esophagus	4	10	--	--	3	4	--	--
Stomach	6	13	--	--	5	10	--	--
Small intestine	2	4	--	--	1	3	--	--
Colon	34	43	--	--	20	20	--	--
Rectum	12	16	--	--	7	6	1.16	NS
Liver	3	3	--	--	3	3	--	--
Gallbladder, pancreas	13	18	--	--	12	14	--	--
Other digestive	3	2	1.17	NS	3	2	1.17	NS
Nasal, larynx	1	2	--	--	0	1	--	--
Trachea, bronchus, lung	10	10	--	--	8	10	--	--
Other respiratory & intrathoracic organs	0	2	--	--	0	1	--	--
Hematopoietic & reticuloendothelial systems	15	16	--	--	11	12	--	--
Bone, connective tissue, skin	16	25	--	--	4	7	--	--
Breast	164	160	1.03	NS	60	49	1.23	NS
Genitourinary	155	174	--	--	52	51	1.02	NS
Bladder	14	7	2.08	.05	6	3	2.57	NS
Eye, nervous system	9	14	--	--	9	12	--	--
Endocrine glands	17	11	1.48	NS	3	2	1.17	NS
Ill-defined	0	0	--	--	0	0	--	--
Lymph nodes	16	15	1.09	NS	8	10	--	--
Unknown	15	16	--	--	13	15	--	--

*Utah Cancer Registry Data by County, 1966-1975.

Table VI-29. *AGE-ADJUSTED INCIDENCE AND MORTALITY RATES FOR WHITE MALES IN UTAH**

CAUSE OF DEATH Site of Neoplasm	INCIDENCE RATES		Relative Risk	Level of Signif.	MORTALITY RATES		Relative Risk	Level of Signif.
	Utah County	Weber County			Utah County	Weber County		
Oral, pharynx, esophagus	45	49	--	--	19	24	--	--
Stomach	17	17	--	--	14	15	--	--
Small intestine	2	3	--	--	0	2	--	--
Colon	36	50	--	--	22	30	--	--
Rectum	17	21	--	--	10	13	--	--
Liver	2	0	--	--	2	0	--	--
Gallbladder, pancreas	20	15	1.22	NS	19	15	1.17	NS
Other digestive	3	1	2.85	NS	2	1	1.17	NS
Nasal, larynx	10	19	--	--	7	7	--	--
Trachea, bronchus, lung	81	79	1.00	NS	76	71	1.05	NS
Other respiratory & intrathoracic organs	2	3	--	--	2	3	--	--
Hematopoietic & reticuloendothelial systems	22	22	--	--	17	19	--	--
Bone, connective tissue, skin	20	14	1.40	NS	7	5	1.40	NS
Breast	3	3	--	--	1	1	--	--
Genitourinary	161	145	1.10	NS	65	75	--	--
Bladder	24	37	--	--	11	20	--	--
Eye, nervous system	15	9	1.75	NS	11	7	1.67	NS
Endocrine glands	5	3	1.47	NS	2	0	--	--
Ill-defined	0	0	--	--	0	0	--	--
Lymph nodes	27	17	1.62	.05	19	11	1.73	NS
Unknown	24	19	1.27	NS	23	18	1.25	NS

*Utah Cancer Registry Data by County for 1966-1975.

D. RESULTS OF ANALYSIS

1. Basis for Initial Causal Hypotheses

The data from Johnstown, Pennsylvania, were analyzed in order to develop hypotheses relating the steel industry emissions, specifically airborne emissions, to increased community mortality. We found deaths from several causes to be significantly elevated; these are listed in Table VI-30.

Table VI-30

CAUSES OF DEATH WHICH ARE SIGNIFICANTLY[†] ELEVATED IN THE EXPOSED AREAS OF THE JOHNSTOWN AIR BASIN

CAUSE OF DEATH (ECI #); ICDA Code*	Significantly Elevated?	
	WM	WF
Oral cancers (2);140-150	Yes	Yes
Stomach cancers (3);151	Yes	No
Peritoneal & unspecified digestive cancers (8);158-159	No	Yes
Respiratory cancer (9);160-163	Yes	Yes
Breast & other cancers (12);170-174,190-199,210-239	Yes	Yes
Leukemia (13);200-209	Yes	No
Endocrine, nutritional, metabolic & blood disease (14);240-289	Yes	No
Hypertensive disease (17);400-404	Yes	No
Cardiovascular diseases (18);410-458	Yes	Yes
Cerebrovascular diseases (19);430-438	Yes	No
Respiratory diseases (20);460-486,500-508,510-514,516-519	No	Yes
Bronchitis, emphysema & asthma (21);490-493	Yes	No
Diseases of liver, gallbladder & pancreas (27);570-577	Yes	No
Diseases of urinary system (28);580-599	No	Yes

[†] $p < 0.05$

*ICDA Codes (8th Revision)

Five of the six air sampling stations in the air basin were within the exposed area. Data from these five sites were averaged to give a "mean" pollution level in the exposed area. The data from the Westmont site were used as representative values for the unexposed area on the recommendation of State officials and due to the lack of air quality data for control areas (a common problem throughout the nation). These data are shown in Table VI-31.

Table VI-31

AIR QUALITY DATA FOR EXPOSED AND UNEXPOSED AREAS OF JOHNSTOWN SITE

POLLUTANT	UNITS	LEVELS	
		Exposed	Unexposed
BaP	ng/m^3	10.4	2.3
BSO	μg/m^3	5.7	2.2
Lead	μg/m^3	.890	.193
Cadmium	μg/m^3	.005	.003
TSP	μg/m^3	101	42
Sulfate	μg/m^3	16.0	11.7

Six of the fourteen causes of death which were elevated (as shown in Table VI-30) were cancers. As the air quality data show, the levels of BaP and BSO in the exposed area are two and a half to almost five times higher than in the unexposed area. The level of particulates was also elevated two and a half times in the exposed area. BaP is a known potent carcinogen. The composition of the BSO may be varied, depending on the coal used for coking, the amount and type of oily scrap fed through the sintering plant, etc. As discussed earlier in this chapter, however, many known hazardous and carcinogenic organics are present in the steel industry emissions, particularly from coking processes. Particulates have also been shown to be associated with stomach cancer.

Sulfur dioxide, particulates, organics, and several trace metals have been implicated in respiratory diseases including bronchitis. These pollutants are also elevated in the exposed area. The liver and urinary diseases have been linked with exposure to lead, other trace elements, and sulfur dioxide. The hypertensive, cardiovascular, and cerebrovascular diseases have been linked to elevated cadmium levels; although the cadmium elevation we show between exposed and unexposed may not be great enough to explain the elevations of these diseases found in Johnstown.

From the above analyses, we can postulate the following hypotheses:

For males and females:

- Oral, respiratory, breast, and genitourinary cancers caused by high levels of BaP and other organics.
- Cardiovascular disease caused by excess cadmium.

For males only (possibly occupational):

- Stomach cancers and leukemia caused by high levels of BaP and other organics plus particulates.
- Endocrine, metabolic, and blood diseases from elevated trace metal concentrations.
- Hypertensive and cerebrovascular disease caused by excess cadmium.
- Bronchitis, emphysema, and asthma caused by elevated SO_2 levels.
- Liver disease caused by exposure to organics and trace elements.

For females only:

- Some digestive cancers caused by exposure to organics.
- Respiratory diseases due to SO_2 exposure.
- Urinary diseases due to trace metal exposure.

Nearly all causes of death are elevated in the female population at risk compared to the control population. Where deaths in the control populations exceed those in the population at risk, the differences are negligible. Conversely, the population at risk shows several causes of death which are more frequent than in the control population and which have more than twofold relative risks. These include cancers of the oral cavity, pharynx and esophagus, respiratory system, peritoneum and retroperitoneum and other nonspecific digestive system sites, and diseases of the urinary system.

In males, only two causes of death are more frequent in the control population than in the population at risk, and both can be reasonably attributed to employment in the coal mining industry. In light of previous studies it is not surprising that respiratory cancer rates are higher in the population at risk; an indication of probable occupationally related disease. The same reasoning would seem to apply to other respiratory and cardiovascular diseases. Two relative risks achieve statistical significance and exceed 2.0. These are for cancers of the respiratory tract and for hypertensive disease.

2. *Hypothesis Test Results*

The hypotheses (p. VI-60) were tested using the data from the Provo site. After analyzing the mortality data for Provo, we found that several causes of death were elevated, but generally not significantly. These are listed in Table VI-32. Since we analyzed data for eight communities exposed to the steel emissions at this site, elevated rates are based on the number of communities which showed elevations as well as the number which had significantly elevated rates.

Table VI-33 shows the results of comparing the elevated causes of death in Johnstown with those causes of death which were elevated in Utah. We found that there were, indeed, causes of death which were elevated in Johnstown that were also high in Utah. Specifically, deaths from breast and other cancers were high in both males and females in Johnstown and also in Utah. Looking at the cancer registry data from Utah showed this elevation to be due, at least at a county level, to excess deaths from neoplasms of the breast and endocrine glands in females and from neoplasms of the nervous system and endocrine glands in males.

Table VI-32

CAUSES OF DEATH WHICH ARE ELEVATED IN THE UTAH COMMUNITIES NEAR THE GENEVA WORKS

CAUSE OF DEATH (BCI #); ICDA Code*	NUMBER OF COMMUNITIES			
	White Males		White Females	
	# Elevated	# Signif.	# Elevated	# Signif.
Pancreatic cancer (7);157	-	-	3	1
Respiratory cancer (9);160-163	3	0	-	-
Breast & other cancers (12);170-174,190-199,210-239	7	2	6	0
Leukemia (13);200-209	3	0	3	0
Endocrine, nutritional, metabolic & blood diseases (14);240-289	4	3	4	0
Cardiovascular diseases (18);410-458	-	-	4	0
Cerebrovascular diseases (19);430-438	-	-	6	2
Respiratory diseases (20);460-486,500-508,510-514,516-519	-	-	3	0
Skin, musculoskeletal & ill-defined diseases (31);680-739,780-796	-	-	4	3
Congenital anomalies (32);740-759	4	1	-	-

*ICDA Codes (8th Revision)

Table VI-33

ELEVATED CAUSES OF DEATH IN JOHNSTOWN WHICH WERE ALSO ELEVATED IN UTAH

ELEVATED CAUSES OF DEATH IN JOHNSTOWN	UTAH RESULT
WHITE MALES & FEMALES:	
Cancer, oral, pharynx & esophagus	Not elevated
Cancer, respiratory organs	Elevated only in WM (not significantly)
Breast & other cancers	Elevated in WM & WF (significant only in WM)
Cardiovascular diseases	Elevated only in WF (not significantly)
WHITE MALES:	
Stomach cancers	Not elevated
Leukemia	Elevated (not significantly)
Endocrine, nutritional, metabolic & blood diseases	Significantly elevated
Hypertensive disease	Not elevated
Cerebrovascular diseases	Not elevated
Bronchitis, emphysema & asthma	Not elevated
Diseases of liver, gallbladder & pancreas	Not elevated
WHITE FEMALES:	
Peritoneal & unspecified digestive cancers	Not elevated
Respiratory diseases	Elevated (not significantly)
Diseases of urinary system	Not elevated

Respiratory cancer was significantly higher in Johnstown, in both males and females. In Utah it was higher only in males, and not significantly higher there. This may be due to decreased exposure to BaP and BSO in Utah, where the community levels are much lower. If this is true, the increased rate in males might be due to occupational exposure to higher levels. Another important factor in this instance is the complicating influence of tobacco use. In the Utah communities studied, 60% to 80% of the population are Mormon. Since most Mormons do not smoke, the rates in the exposed and control populations could be greatly reduced due to the factor. This, then, could indicate that the combination of smoking and high BaP levels in Johnstown causes the excessive deaths.

Cardiovascular disease, which was elevated in both men and women in Johnstown was elevated in women only in Utah, and not significantly. This might suggest a different etiology than previously presented, since our initial hypothesis in Johnstown was based on cadmium, and the cadmium levels were only slightly elevated in the exposed areas. Possibly the linkage of cardiovascular heart failure with respiratory dysfunction (see Etiology Section in Chapter VII, Bituminous Coal Mining Case Study) is the cause for elevation of the disease in Johnstown.

In Johnstown, deaths from endocrine, metabolic, and blood diseases were significantly elevated in males only. This was again true in Utah where females showed rates that were elevated, but not significantly so. This may be occupational exposure, or it could reflect the difference in reaction to the relevant pollutant between males and females.

Leukemia was significantly elevated in males in Johnstown and elevated, but not significantly so, in Utah. The same was true for respiratory diseases in females.

In general, the effects of the steel industry on Utah communities would be expected to be less than in Johnstown due to the lower levels of pollutants. Figure IV-8 shows the difference in BaP levels with respect to distance from the plants in Johnstown and in Utah. As can be seen from the figure, concentrations at the same distance from the plant are much higher in Johnstown, due to both the amount of plant emissions and the geography. The difference in pollutant levels should have a profound effect on community health and is the probable reason why we found fewer causes of death elevated in Utah than in Johnstown.

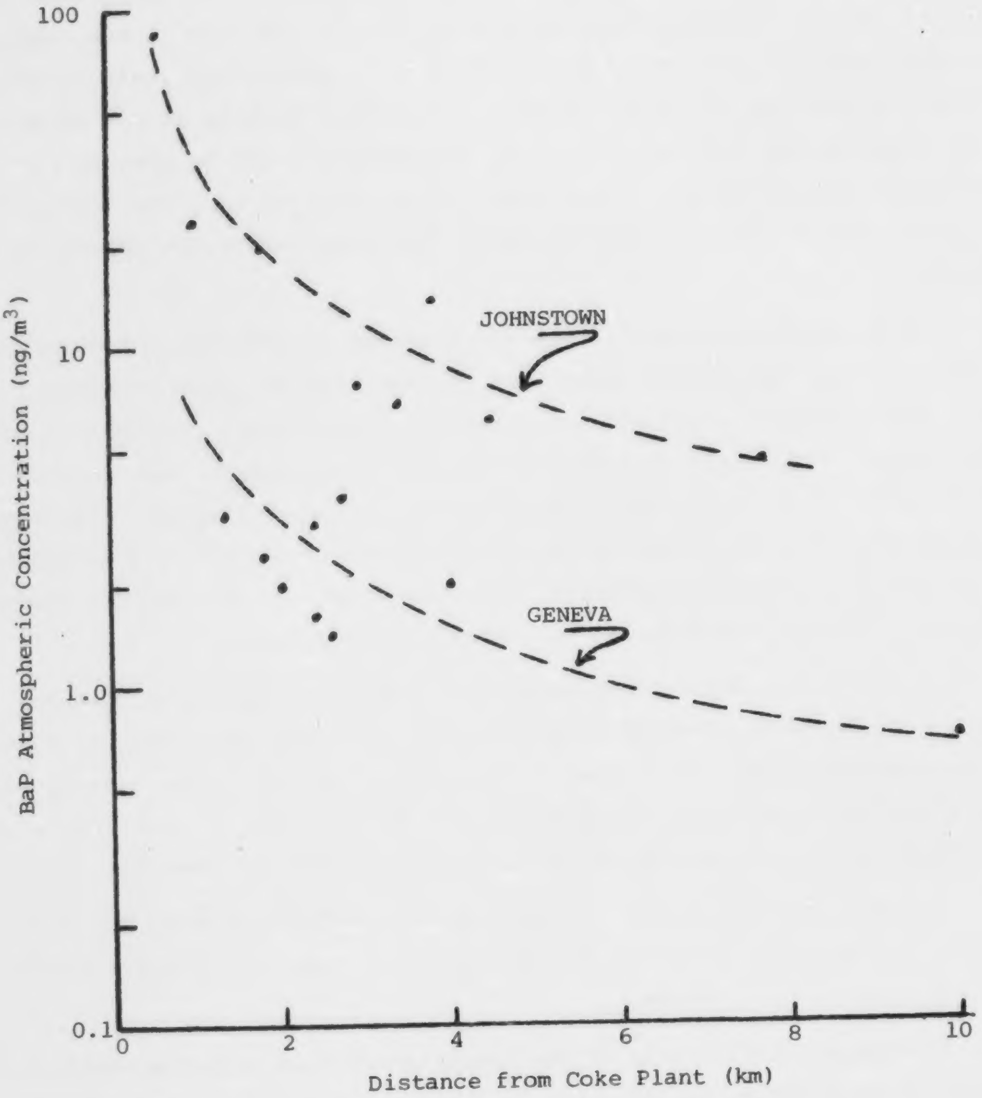

Figure VI-8

Atmospheric Concentrations of BaP: Johnstown, Penn., and Geneva, Utah

3. *Methodological Findings*

One of the primary methodologies developed in this study was the use of computerized mortality and cancer registry data. Given the scope of the project and the time limitations, use of hard copy data was not possible. The data available on tape had their own limitations—lack of occupational, length of residence, and religious (important in Utah) information. Another factor was residency reporting. Giving residency by town rather than by census tract or enumeration district was fine in some cases; however, in Johnstown we would have liked to analyze the areas in the City of Johnstown closer to the Franklin Works separately from those areas to the south. In most cases, more detailed residency reporting would have made dose-response analyses possible.

The residency information on the Utah Cancer Registry tape was particularly disappointing. Only two years of data with detailed residency information were available, and this was by zip code which is not easily converted to populations. We were forced to analyze the bulk of the registry data by county, which is quite limiting for an analysis of this sort.

After deciding how to prepare the basic data, i.e., how to group ages, how to age-adjust and for which ages, which demographics to examine, etc., we had to determine which statistical methods were valid as applied to our data. Several methods were considered, including the Cochran test, Chiang's method, and tests using the Poisson distribution. The method finally decided upon was the Mantel-Haenszel χ^2 test.

Another methodology developed in this study involved the use of interpretation of aerial photography. Barry Evans of EPIC assisted us in this by tracing the presence, growth, and change of polluting sources in Johnstown from 1939 to the present (see Appendix I). Besides the data presented in the appendix, topographic maps were prepared with overlays displaying polluting sources and residential areas for each of the years examined. This graphic representation of the historical residential and industrial patterns was very useful to us in analyzing the data from this site.

Any analysis of the kind we performed depends on the availability of air quality data for the pollutants of interest. We found that these data varied greatly, not only from state to state but also from test site to control site. Pennsylvania had data for criteria pollutants plus heavy metals, ions, BaP, BSO, etc., for several years in the Johnstown Air Basin. For the rest of Cambria County, however, there were no data. Utah data, except for a special study done by EPA, were strictly for criteria pollutants. Here again, data for the control site were scarce.

One of the points to be developed in this analysis was how our data, analyzed on a town level, compared with the previously done regression data which had been analyzed on a county level. Table VI-34 shows how the basic data from each method compares in Johnstown. An important factor in any analysis of this sort is sufficient population at risk. This criterion is more easily met using county data, although the steel industry investigation had sufficient populations on a town level also. If the population immediately surrounding the source is small, the county unit may not show increased mortality. Conversely, factors other than the source of pollution studied in the county might increase countywide mortality rates. Another important consideration is a possible development of a dose-response trend as exposure levels decrease with distance from the source.

Table VI-34. AGE-ADJUSTED MORTALITY RATES, WHITE FEMALES (ages 35-74, per 100,000, annualized)

CAUSE OF DEATH		1965-1975 Cambria County			1968-1972 SSI Data Cambria County	
SSI #	ECI #	County	Exposed	Unexposed	County	National
1	1-34[a]	1,102	1,273	988	3,617	883
4	2-13	274	278	231	252	253
5	2*	2	6	0	1	4
6	3-8*	69	63	60	75	63
7	3	10	11	7	10	8
8-9	4-5	33	43	34	43	35
10	7	8	9	9	5	12
11	6,8*	13	20	10	15	9
12	9	20	26	12	16	26
16,17,19	10	51	44	49	43	45
18	11	4	3	2	3	3
15,20,23	12	102	111	85	85	90
21,22	13	27	25	23	23	22
24-26	14	66	61	76	58	34
27	16-18*	589	708	528	494	398
28	16	34	36	27	25	15
29	17	22	27	19	6	9
31-37	18	534	645	482	500	384
34	19	111	127	105	107	84
38	20-22	37	52	32	34	37
40	21	11	12	9	8	11
46	23-27	54	60	47	62	45
50	28-29	13	19	9	34	23

[a] Except 19.

*Some difference in ICDA numbers, SSI vs. ECI.

4. *Evaluation of Investigative Approach*

One of the important evaluations which must be made from this study concerns the value of the original regression approach for the steel industry. The steel industry indicated by the regression was a very broad SIC code containing many sources and types of pollutants. This makes isolating the pertinent factor(s) with health effects difficult. Another drawback, particularly in the steel industry, was the industrial clutter found in counties with the steel industry. Our findings in Utah seem to indicate that there is a minimum pollutant level, below which few health effects are seen. In steel, it is difficult to find counties with high pollutant levels and little industrial clutter. Where other polluting sources are present, the matter of implicating steel industry emissions is much more complicated.

The approach we selected for studying industrial activity and causally related community disease was a two-phase effort, first hypothesis development and subsequent hypothesis testing. Its implementation in Johnstown and Provo brought to light several possible limitations of its use. Site selection is a problem. If one selects a test site to avoid other sources of pollution, as we selected Provo, it is possible that the industrial activity and the resultant pollution levels are too low to produce a measureable effect. Thus, care must be taken to select sites with sufficient industrial activity and emissions. This may force the investigation to the larger metropolitan areas where general urban pollution becomes a problem.

There are, however, several strong points in the approach we selected. First, it was an objective approach, at least within the limits of data availability. It was also industry-specific, that is, defining exactly what processes and, therefore, what pollutants were present at the sites studied. We studied two fully integrated steel mills and characterized emissions by processes. Finally, the use of data more specific than county data allowed us to see mortality patterns with respect to actual pollution levels at the sites.

E. *CONCLUSIONS AND RECOMMENDATIONS*

Some of the hypotheses that were developed in Johnstown were supported by the Utah data. Notable among these, especially since it applied to both males and females, was malignant neoplasms caused by increased exposure to organic emissions, especially BaP. Even more convincing, perhaps, is the fact of this finding even though exposure levels in Utah were low and, perhaps due to religious practices, cancer mortalities are low.

Of course, some hypotheses developed in Johnstown did not seem to hold up well in Utah. These could also be explained by religious and exposure differences, by climatological and geographic differences, or by ethnic and demographic differences. It seems likely that there simply was not a long or high enough exposure in Utah to cause noticeable health effects.

Several aspects of this study could be expanded to give a greater insight into exactly what emissions and at what levels are health hazards. Determining occupation, religion, and length of residence of the decedents, while requiring a time-consuming study of hard copy data, would do much to characterize mortality patterns.

Obtaining more complete air quality data is also important. Since the parameters we are interested in are often not measured, this might involve actual sampling and assay, especially in designated control areas where the present monitoring networks have no samplers.

Finally, a detailed, in-depth study of a large industrial city with high industrial activity with respect to steel, such as Pittsburgh or Detroit, would give us a large enough population at risk to make a detailed analysis of mortality patterns, especially with regard to a dose-response relationship for specific pollutants.

Chapter VII

BITUMINOUS COAL MINING CASE STUDY

A. *INTRODUCTION*

This chapter documents the field investigation of the association between bituminous coal mining and respiratory/cardiovascular diseases. This association was clearly the strongest correlation found by the multiple regression analysis of county industry and mortality data [1]. It occurred in both males and females and in both county samples, where each sample contained roughly half of the 3,000 U.S. counties. The correlation was convincingly validated by the top 10 county matched-control test. Most of the component diseases that comprise respiratory and cardiovascular diseases were found to be correlated with the industry. Because of the unusual strength of the correlation, particularly in females, we felt that it was the most likely of all correlations to represent a true cause-and-effect association between an industry and community disease.

The listing below, extracted from the Standard Industrial Classification (SIC) Manual [2] describes the several components of the three-digit SIC code (SIC 121) correlated with respiratory and cardiovascular diseases. The various diseases comprising respiratory/cardiovascular diseases are listed in Table VII-1.

Group No.	**Industry No.**	
121		**BITUMINOUS COAL AND LIGNITE MINING**
	1211	**Bituminous Coal and Lignite** **Establishments primarily engaged in producing bituminous coal or lignite or in developing bituminous coal or lignite mines. This industry includes underground mining, auger mining, strip mining, culm bank mining, and coal cleaning, crushing, screening, and sizing plants, whether or not operated in conjunction with the mines served.**

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[1] System Sciences, Inc., *Investigation Into the Industrial Correlates of Environment-Related Mortality*, 1976.

[2] Office of Management and Budget, *Standard Industrial Classification Manual*, 1972.

1213 Bituminous Coal and Lignite Mining Services

Establishments primarily engaged in performing bituminous coal and lignite mining services for others on a contract, fee, or similar basis. Establishments which have complete responsibility for operating mines for others on a contract, fee, or similar basis are classified according to the product mined rather than as bituminous coal and lignite mining services.

Table VII-1

CORRELATED CAUSES OF DEATH—RESPIRATORY AND CARDIOVASCULAR DISEASES

Respiratory/Cardiovascular Diseases Components	ICDA Number (8th Rev.)
Respiratory Diseases	
Bronchopneumonia	485
Acute interstitial pneumonia	484
Emphysema	492
Asthma	493
Acute nasopharyngitis	460
Acute sinusitis	461
Acute laryngitis and tracheitis	464
Acute pharyngitis	462
Acute tonsillitis	463
Acute upper respiratory infection of multiple or unspecified sites	465
Acute bronchitis and bronchiolitis	466
Influenza, unqualified	470
Influenza with pneumonia	471
Influenza with other respiratory manifestations	472
Influenza with digestive manifestations	473
Viral pneumonia	480
Pneumococcal pneumonia	481
Other bacterial pneumonia	482
Pneumonia, unspecified	486
Hypertrophy of tonsils and adenoids	500
Peritonsillar abscess	501
Chronic pharyngitis and nasopharyngitis	502
Chronic sinusitis	503
Chronic laryngitis	506
Other diseases of upper respiratory tract	508
Spontaneous pneumothorax	512
Pulmonary congestion and hypostasis	514
Other pneumoconioses and related diseases	516
Bronchiectasis	518
Other diseases of respiratory system	519
Cardiovascular Diseases	
Acute myocardial infarction	410
Other acute and subacute forms of ischemic heart disease	411
Chronic ischemic heart disease	412
Angina pectoris	413
Asymptomatic ischemic heart disease	414
Pulmonary embolism and infarction	450
Phlebitis and thrombophlebitis	451
Portal vein thrombosis	452
Other venous embolism and thrombosis	453
Varicose veins of lower extremities	454
Varicose veins of other sites	456
Noninfective disease of lymphatic channels	457
Other diseases of circulatory system	458

To investigate the relationship between bituminous coal mining and respiratory/cardiovascular diseases in the community, field studies were conducted at two separate sites: Wyoming County, West Virginia (see Figure VII-1) and the bituminous coal mining region in southern Illinois consisting of a 5-county area (see Figure VII-2). Wyoming County was studied first, for the purpose of developing a plausible causal hypothesis. The possible sources of pollution in the county were investigated, including their history, magnitude, distribution with respect to the population, and possible toxic effects. Based on these data and intercounty and intracounty mortality patterns, plausible hypotheses were constructed.

Testing of the hypotheses was accomplished by two methods: by a morbidity study in Wyoming County and by a mortality study at the southern Illinois site. The latter site was selected principally because of industrial characteristics befitting the hypothesis being tested and the ready availability of mortality data.

B. *HYPOTHESIS DEVELOPMENT*

1. *Approach*

To attempt to construct a plausible causal hypothesis for the association between bituminous coal mining and respiratory/cardiovascular disease, an investigation was carried out in Wyoming County, West Virginia, consisting of five separate but related research efforts. The first two, which were conducted more or less in parallel, consisted of (1) a thorough study of the coal mining industry in general, and in Wyoming County in particular, to fully characterize the mining-related sources of pollution in the community, and (2) etiology research of respiratory and cardiovascular diseases in coal miners and coal mining communities to identify medical evidence of connections between toxic substances from mining operations and disease. At this point, several candidate hypotheses were postulated involving sepecific sources of toxic agents produced by the mining operations. These hypotheses were examined in light of the next two research efforts to determine which of the candidates were plausible, giving consideration to the site-

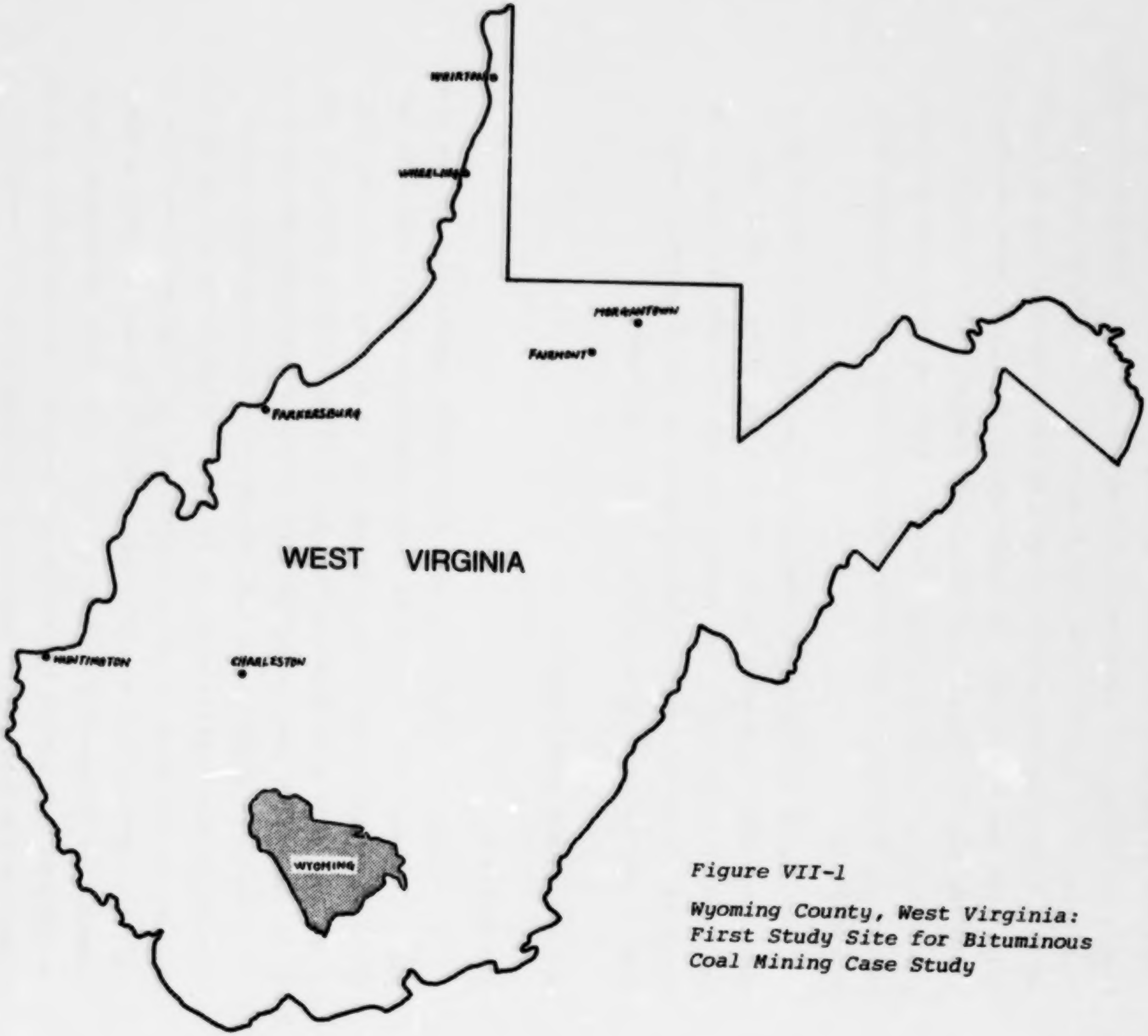

Figure VII-1

Wyoming County, West Virginia: First Study Site for Bituminous Coal Mining Case Study

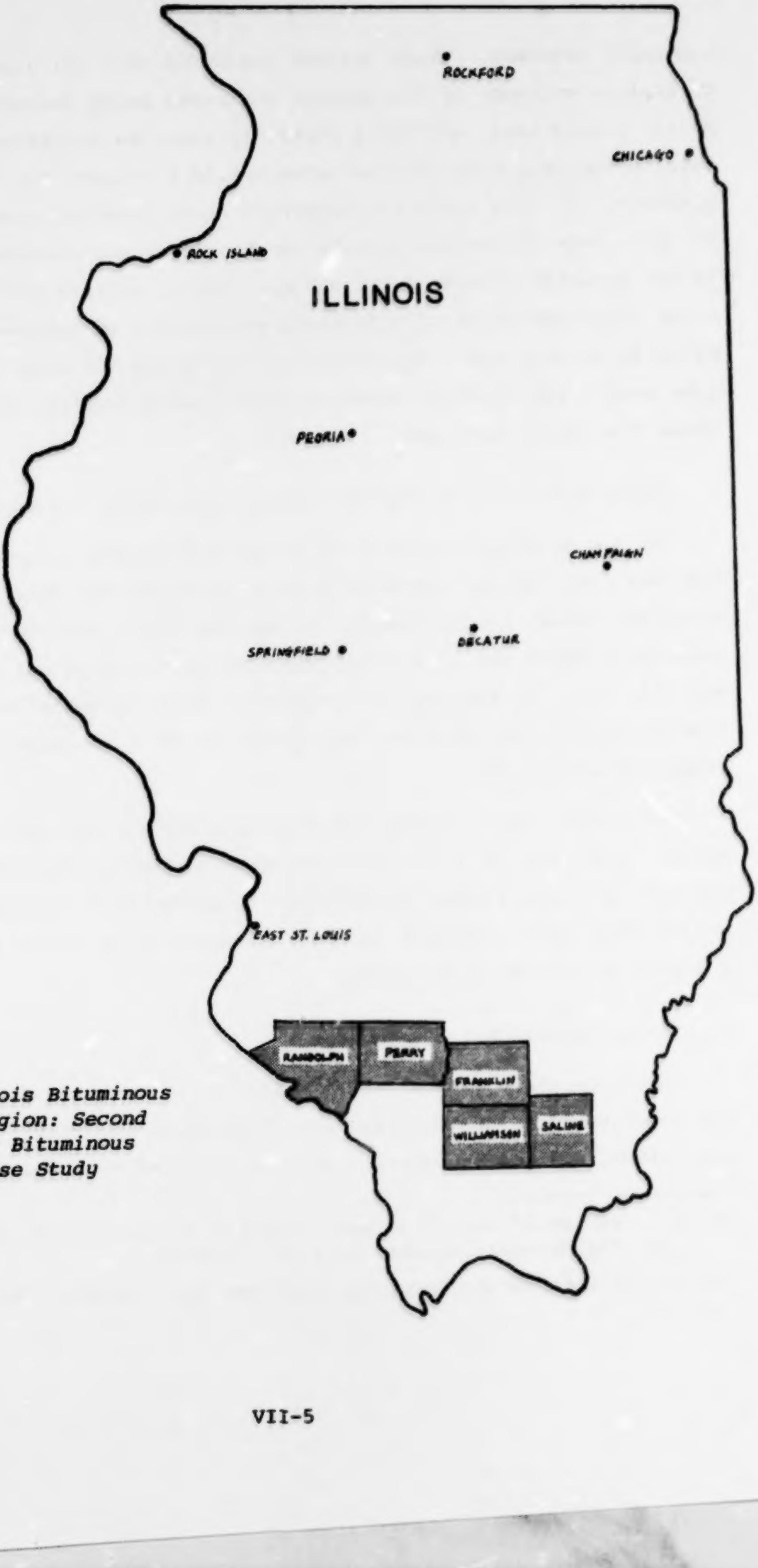

Figure VII-2

Southern Illinois Bituminous Coal Mining Region: Second Study Site for Bituminous Coal Mining Case Study generated evidence. These efforts consisted of: (3) a pathway analysis to explore evidence of the suspect toxicants being transported to community populations, and (4) a mortality study to determine if the disease-specific mortality patterns provided support for any of the hypotheses. At this juncture, questions still remained regarding the possible role of certain factors (particularly socioeconomic factors) in the industry-disease relationship. Hence, a fifth effort was undertaken which consisted of a detailed examination of intercounty disease patterns in Appalachia and elsewhere, using the SSI county mortality data base. The sections immediately following describe the results of these five investigations.

2. *Characterization of the Bituminous Coal Mining Industry*

The two principal operations in the coal mining industry are mining and coal preparation. Wyoming County, which was the third highest coal-producing county in the country during the 1950's and 1960's, has had as many as 90 mines and 15 coal preparation plants (tipples) operating at one time [3]. In general, the number of mines is decreasing and their size is increasing; however, the county is still a highly ranked coal-producing county [4].

The operations of mining and coal preparation are each described below. Following this is a discussion of the environmental pollutants produced by these mining operations. In addition to mining and coal preparation, coal transportation is discussed as an operation which is a source of community pollution.

a. *Mining Operations*

There are three general methods used to mine coal: surface, augur, and underground. The technique used depends on the seam's accessibility, width, and depth. Strip mining is employed when the seam is near the surface. The auger technique is used on seams exposed on hillsides, when the seam is narrow, or the overburden is unstable for underground mining. Auger mining involves a large auger drill (16 to 84 inches in diameter) that bores horizontally into the coal seam where exposed. Underground mining is classified by the mine's access to the seam. Drift mines enter the coal directly on exposed hillsides. A slope shaft angles down from the mine entrance to the coal seam. When the access is a vertical shaft to a seam far below the surface, it is called a shaft mine.

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[3] U.S. Bureau of Mines and West Virginia Geological and Economic Survey, *The Mineral Industry of West Virginia*,

[4] Mining Information Services, *Keystone Coal Industry Manual* series,

In Wyoming County most mines (~90%) are the underground type. Surface mining in the area (~7%) is the contour technique which follows a coal seam outcropping around a mountainside. Because seams are usually horizontal, the overall effect of a mining operation is a contour line around a mountain. Auger mining (~3%) usually follows the contour strip mining [4].

Coal is transported from the mines by train, truck, or conveyor to preparation plants or directly to a consumer. When coal preparation plants (tipples) are close at hand, conveyor systems carry coal directly to the tipple raw-coal storage. Small operations usually truck coal out, while large mines may load directly onto a train.

b. *Coal Preparation Operations*

Coal preparation plants, commonly called tipples, perform three functions: the crushing of coal lumps to specific sizes, the separation of impurities (dirt, rock, etc.), and the removal of excess moisture.

A series of crushers, grinders, and screens are used to reduce and separate the coal to uniform sizes. The finer the coal is crushed, the more impurities, which are physically trapped in the coal, can be removed in the process. The crushing and screening processes are for the most part enclosed, resulting in limited fugitive coal dust emissions.

Impurities are separated by two cleaning processes: washing with water and specific-gravity separation. Large coal sizes are sprayed with water to rinse off clinging dirt particles. The separation processes for smaller sizes of coal exploit the differences in specific gravity between coal and impurities (coal is lighter). Usually water and sometimes air is used as the medium. Effluents and emissions from these processes include dirt, rock, and coal particles.

Drying of the coal is done by natural draining, screening, centrifugation, filtering, and thermal drying. Effluents from these processes again contain coal, rock, and dirt particles. Emissions are generally limited due to the wet nature of the material. However, in the case of thermal drying, large quantities of coal dust are entrained by the hot flue gases used for drying the coal. The majority of coal preparation plants in the United States use thermal dryers. Table VII-2 lists the types and numbers of thermal dryers in the United States.

Table VII-2

*THERMAL DRYERS IN THE UNITED STATES IN 1973**

Type	Number
Fluidized Bed	66
Multi-louver	16
Rotary	36
Screen	12
Suspension or Flask	31
Vertical Tray & Cascade	1

**Source:* EPA, *Coal Preparation Environmental Engineering Manual*, Publ. No. EPA-6002-76-138, May 1976.

Open coal storage piles are not actual processes of tipples but are associated with them. Uncovered piles of coal are sources of fugitive emissions and leachate from rainwater. Also, the handling of coal by trucks or conveyors around tipples generates fugitive coal dust emissions.

In Wyoming County there are five thermal dryers currently in operation; Figure VII-3 is a map of Wyoming County showing the location of these tipples.

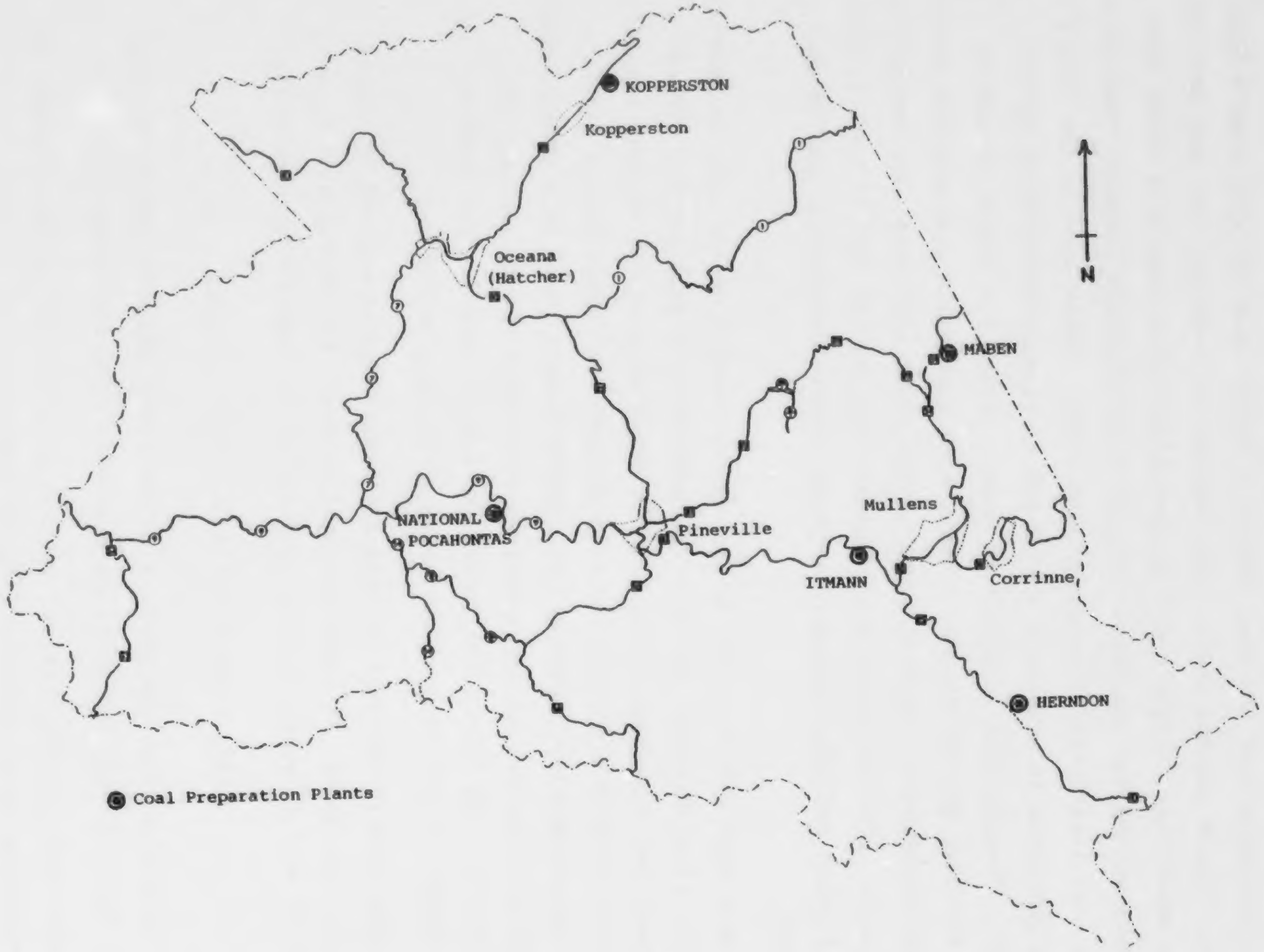

Figure VII-4. Map of Wyoming County: Location of Coal Preparation Plants

c. *Environmental Pollution from Mining Operations*

Pollutants from the coal industry include mine seepage, tipple solid waste, tipple process water, coal combustion products, and coal particulate emissions. Active and abandoned mines often seep acid water into surface waters and aquifers. Tipples dump the waste material separated from coal into large piles around the plant. Water used in the separation processes is sometimes acidic and is usually released to surface water. Because coal is burned to supply energy for tipple processes and hot flue gas for thermal dryers, coal combustion products are emitted to the atmosphere. Burning tipple waste piles also emit coal combustion products. Coal particles are entrained and emitted into the air by mine ventilation systems, transport systems, tipple operations, and thermal dryers.

Acid Mine Drainage—Due to the sulfur content of the coal in Wyoming County, it is possible for mine drainage to be highly acidic. If the drainage has a pathway to the potable water supply, the various minerals that can be solubilized by the acid en route can constitute a significant health problem. However, in Wyoming County there is a considerable amount of limestone in the substrata which will neutralize the acid drainage to some extent and reduce the health hazard. The chemistry of acid formation and neutralization is postulated as follows.

Once pyritic material in coal and overburden is exposed to oxygen (in air) and water (in vapor or liquid phase), the sulfur will be oxidized to sulfite which in turn is hydrated to form sulfuric acid [5]:

$$FeS_2 + H_2O + 7/2\ O_2 \longrightarrow FeSO_4 + H_2SO_4$$

Acid formation drops the pH to 2 to 4.5 which tends to solubilize minerals (iron, manganese, etc.) at a greater rate than normal.

Upon reaching the surface, several reactions occur which result in the formation of bright-orange ferric hydroxide precipitate:

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[5] Grim, E. C., and R. D. Hill, *Environmental Protection in Surface Mining of Coal*, EPA-670/2-74-093, EPA, Cincinnati, Ohio, October 1974.

$$2\ FeSO_4 + 1/2\ O_2 + H_2SO_4 \longrightarrow Fe_2(SO_4)_3 + H_2O$$

$$Fe_2(SO_4)_3 + 6\ H_2O \longrightarrow 2\ Fe(OH)_3 + 3\ H_2SO_4$$

Neutralization of acidic mine drainage occurs as native limestone ($CaCO_3$) reacts with sulfuric acid to form carbonic acid and bicarbonate:

$$H_2SO_4 + CaCO_3 \longrightarrow CO_2 + H_2O + Ca^{++} + SO_4^{=}$$

$$CO_2 + H_2O \longrightarrow H_2CO_3$$

$$CaCO_3 + 2\ H_2CO_3 \longrightarrow 2\ HCO_3^- + CO_2 + Ca^{++} + H_2O$$

The end result of neutralized acid mine drainage is an excess amount of minerals and trace elements and excess total dissolved solids, especially sulfates and bicarbonates. These latter compounds can exert laxative effects on humans. Trace metals in sufficient quantity may pose a number of public health threats. High quantities of divalent ions (Ca^{++}, Mg^{++}) cause elevation in water hardness. The public health significance of water hardness is not clear, but researchers have attempted to correlate many chronic diseases, particularly cardiovascular diseases (heart disease, hypertension, and stroke) with low water hardness.

Coal Dust Emissions—Coal dust emissions originate from mine ventilation exhaust, transport vehicles, preparation plant operations (fugitive), and thermal dryers [6].

It is conceivable that the coal dust carried out of underground mines by ventilation systems could create environmental pollution. However, significant amounts of pollution have never been shown to arise from ventilation exhausts. These systems do not actively entrain or heat coal dust; they only move the dust out of the mines. Ventilation shaft locations change over time because they follow the working face of the mine; consequently, pollution from shafts would generally be short term. Also, the exhausts are usually located a considerable distance from the populated areas. Consequently, mine ventilation exhausts are not considered a major air pollution source.

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[6] Seeley Mudd Series, *Coal Preparation*.

Uncovered transport vehicles (railroad cars and trucks) disperse coal dust throughout their routes. Railroad tracks are historically stable, while truck routes vary with mine locations. Accurately defined truck routes for the past 40 years are not available. The magnitude and characteristics of this source of coal dust are extremely difficult to estimate.

Fugitive emissions from tipples occur from the handling, storage, and operations of the tipple. The loading and unloading of ROM and clean coal, respectively, create fugitive emissions. Uncovered conveyors are also sources of windblown dust. Within the plant, the following operations may create fugitive emissions:

- sizing
- prewetting
- dewatering
- mechanical drying

Emissions from thermal dryers typically have gas volumes from 85,000 to 425,000 cubic meters per hour, temperatures of about 93°C, and dust concentrations of 230 to 690 g/m^3 [7]. Table VII-3 gives the characteristics of the five thermal dryers in Wyoming County and shows the total particulate matter emitted from each as compiled by NEDS [8]. All emissions are determined without collectors or controls. Table VII-4 displays the characteristics of particulate emissions, particle size distribution and emission factors; also emission estimates for a 500-ton/hr unit are shown for an 8-hr day. There are $\sim 10^{12}$ respirable particles/m^3 emitted from a fluidized-bed unit with a dust loading of 450 g/m^3

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[7] TRW Systems Group and Resources Research, Inc., *Air Pollutant Emission Factors—Supplement*, DHEW Publ. No. PB-206 923, August 1970.

[8] EPA, *National Emissions Data System (NEDS), Point Source Listings*, EPA, Research Triangle Park, North Carolina.

Table VII-3. *THERMAL DRYER (FLUID-BED TYPES) CHARACTERISTICS: WYOMING COUNTY, WEST VIRGINIA**

PLANT/LOCATION	MAXIMUM RATE (tons of coal dried/hr)	CONTROLS		STACK PARAMETERS				Calculated Particulate Emissions (ton/yr)
		Type of Device	Efficiency for Particulates (%)	Height (ft)	Diameter (ft)	Temperature (°F)	Flow Rate (ACFM × 10^3)	
Keystone #2/ Herndon	300	Centrifugal & Wet Scrubber	99.3	93	5.5	130	130	243
Itmann/ Itmann	500	Centrifugal & Wet Scrubber	99.5	136	9.9	110	142.8	107
Kopperston/ Kopperston	410	Centrifugal & Wet Scrubber	98.0	114	17	130	130	80
Maben/ Maben	150	Centrifugal & Wet Scrubber	99.1	30	4	145	67	24
National Pocahontas/ Mullensville	360	Gravity	85	84	5.7	150	180	67

**Source:* NEDS, reference [8].

(specific gravity of coal ∿1.4). Table VII-5 shows the results of an analysis of the particulate emissions from a thermal dryer [9].

Table VII-4. THERMAL DRYER PARTICULATE EMISSIONS CHARACTERIZATION *

	Typical Particulate Size Distribution by % Weight Less Than Stated Sizes					Uncontrolled Emission Factors (lb particulates/ ton coal dried)	Particulate Emissions[a] (tons/day)	
	2μ	10μ	20μ	40μ	60μ		Total	Respirable
Thermal Dryer Emission Ranges	0.1 to 2.5	0.5 to 10	5 to 42	14 to 50	20 to 70	16 to 25	32 to 50	0.16 to 5

**Source:* Reference [7]. [a]For a 500-ton/hr unit and 1 shift (8-hr)/day.

Table VII-5

*ANALYSIS OF PARTICULATE EMISSIONS FROM A THERMAL DRYER**

ELEMENT	CONCENTRATION (ppmw)[a]	ELEMENT	CONCENTRATION (ppmw)[a]
Be	1	Sr	100
Cd	<<50	Na	300
As	<100	K	1000 to 2000
V	50	Ca	3000
Mn	50 to 100	Si	1.5%
Ni	20 to 30	Mg	1000
Sb	<50	Bi	<10
Cr	30	Co	<10
Zn	<100	Ge	<30
Cu	30	Mo	<10
Pb	<30	Ti	500
Se	--	Te	<100
B	10	Zr	10
F	--	Ba	200
Li	<10	Al	1.0%
Ag	<1	Cl^-	40 to 118
Sn	<50	$SO_4^=$	1040 to 3920
Fe	5000		

**Source:* Reference [9]. [a]Parts per million by weight.

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[9] EPA, *Background Information for Standards of Performance: Coal Preparation Plants*, Vol. I, II, III, Publ. No. EPA 450/2-74-021.

The control of transport vehicle and fugitive tipple emissions is accomplished by enclosing the source. Open storage piles are replaced with silos, conveyors are covered, and crushing and screening units are enclosed with treated vents. Vehicles cover their loads with tarpaulins. Consequently, fugitive emissions are controlled by enclosing the coal and restricting its exposure to wind.

Controls on thermal dryer emissions are primary dry centrifugal recovery and secondary wet collectors. Cyclones collect up to 95% by weight of the entrained particulates [7,10] and return them to the product coal. All secondary controls are of the wet venturi type [10]. High-efficiency, high-pressure drop scrubbers can achieve 99% efficiencies by weight. Relative efficiencies of cyclone and venturi collectors by particle size are shown in Figure VII-4. Typical grain loadings from various types of control equipment are as follows [7]:

Type of Control	Outlet Grain Loading, g/scf
Cyclones (product recovery)	6 -9
Multiple Cyclones (product recovery)	2.5 -3.5
Venturi Scrubber* (pressure drop 5 in. water)	0.1 -0.2
Venturi Scrubber* (pressure drop 20 in. water)	0.02 -0.03

*Following product recovery systems on the thermal dryer effluent.

The efficiency of venturi scrubbers is dependent on particle size distribution, and a large pressure drop is required to remove the smaller particles. Consequently, high power inputs are required for higheffiency collection of respirable particles [11]. Venturi scrubbers were not incorporated at Wyoming County dryers until 1969.

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[10] Nunenkamp, D. C., *Coal Preparation Environmental Engineering Manual*, EPA Publ. No. EPA-600/2-76-138, May 1976.

[11] Vandegrift, A. E., et al., *Particulate Pollutant System Study, Vol. III. Handbook of Emission Properties*, Publication PB-203 522, May 1971.

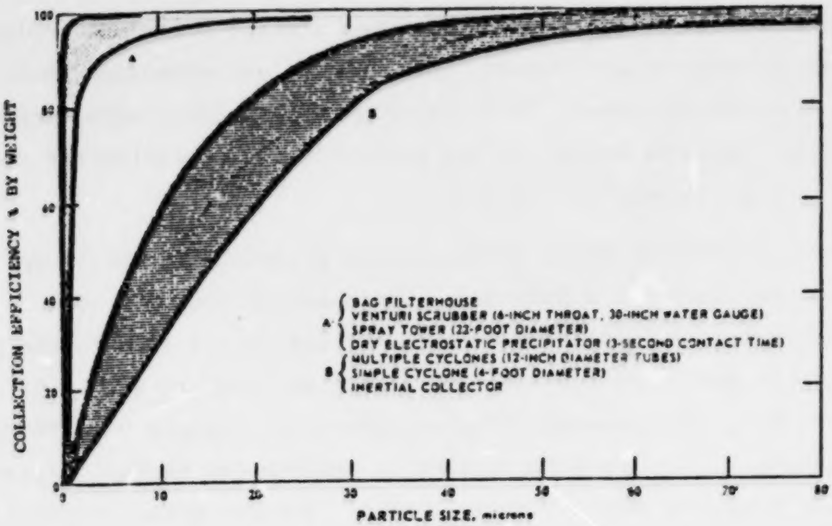

Figure VII-4. Composite Grade (Fractional) Efficiency Curves Based on Test Silica Dust

Coal Combustion Products—The principal sources of coal combustion products are the burning waste (gob) piles and the burning of coal in the thermal dryers to create the flue gas for drying coal. The particulate matter produced by the coal burning in the thermal dryer has been included in the coal dust emission estimates discussed in the previous section. These emissions consist principally of coal dust entrained in the hot air but also include the particulates from the coal combustion process. The gaseous combustion products emitted by the thermal dryer consist principally of nitrogen and sulfur oxides. Because of the relatively low rate of fuel burned (only 5 tons per hour in the largest thermal dryer), the gaseous emissions are quite small and EPA has not found it necessary to set standards for SO_x and NO_x emission controls. Further, NEDS, published by EPA, does not include point source estimates of SO_x and NO_x emissions for thermal dryers (it actually publishes values of zero). EPA has established that the gaseous emissions from thermal dryers are much less than controlled emissions from small coal-burning power plants [9].

Neither actual measurements nor estimates of emission rates were found for burning gob piles. Due to the low coal composition of gob piles and the inaccessibility of oxygen in the piles, actual burning is very slow and emission rates are very low. A count of 15 burning gob piles in Wyoming County was made by the U.S. Bureau of Mines [12].

3. *Etiology*

The part that coal dust plays in chronic respiratory disease in coal miners is well established. It is conceivable, therefore, that coal dust can be the etiologic agent for excessive respiratory disease in coal mining communities. Furthermore, a reasonable case can be made for an indirect connection between cardiovascular disease and coal dust, where the respiratory dysfunction caused by chronic coal dust inhalation accelerates or induces heart disease. The etiological basis for associating coat dust with respiratory and cardiovascular disease is illustrated in Figure VII-5. The etiology is similar to that for coal workers' pneumoconiosis (CWP) in which coal dust is the toxic agent entering the body by the respiratory route, causing chronic respiratory disease which then leads to cardiovascular disease through increased resistance of the pulmonary arterial bed and poor oxygen exchange. In community disease, the exposure would be of long term and low level, in contrast to the shorter term, high-level exposure causing CWP in miners.

The paragraphs below summarize first, the etiology of respiratory disease and second, the etiology of cardiovascular disease associated with coal mining, where the principal etiologic agent is considered to be coal dust. Following this is a brief discussion of the etiology of respiratory and cardiovascular diseases associated with other principal sources of pollution from the coal industry, i.e., acid mine drainage and coal combustion products. Appendix K presents a more thorough treatment of the health effects of the possible etiologic agents associated with mining operations.

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[12] McNay, L. N., *Coal Refuse Fires, An Environmental Hazard*, U.S. Bureau of Mines Information Circular No. IC 8515, 1971.

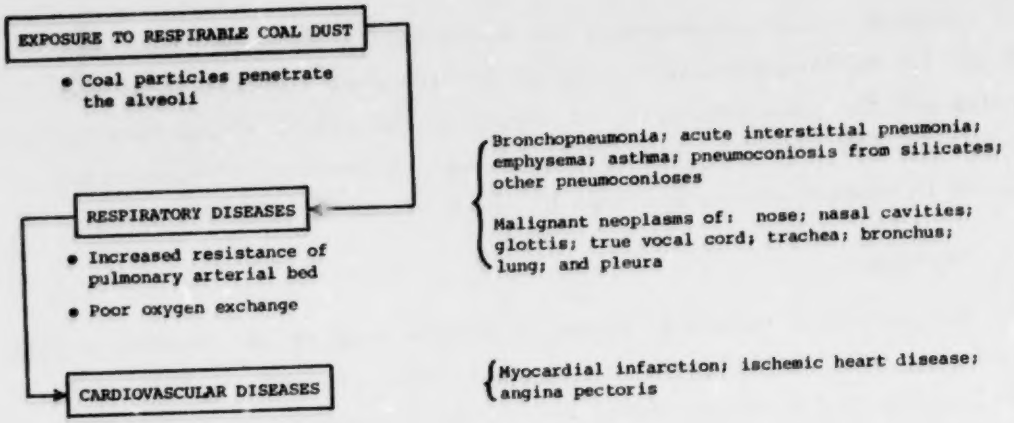
EXPOSURE TO RESPIRABLE COAL DUST
• Coal particles penetrate the alveoli
RESPIRATORY DISEASES
• Increased resistance of pulmonary arterial bed
• Poor oxygen exchange
CARDIOVASCULAR DISEASES
Bronchopneumonia; acute interstitial pneumonia; emphysema; asthma; pneumoconiosis from silicates; other pneumoconioses
Malignant neoplasms of: nose; nasal cavities; glottis; true vocal cord; trachea; bronchus; lung; and pleura
Myocardial infarction; ischemic heart disease; angina pectoris

Figure VII-5. Etiology: Coal Dust Particles

a. *Respiratory Disease Etiology*

Coal miners themselves are predisposed to a variety of illnesses, chiefly respiratory. There are four major occupational respiratory diseases of coal miners:

- Coal Workers' Pneumoconiosis (CWP)—this results from the accumulation of inhaled coal dust in discrete macules in the lung and can be associated with some changes in respiratory function, rarely if ever disabling.
- Progressive Massive Fibrosis (PMF, or "complicated CWP)—this is an advanced, uncommon form of CWP which results in obstructive (emphysematous) and restrictive pulmonary disease and may be life threatening.
- Chronic Bronchitis—this probably represents chronic bronchial irritation from dust, but has not been extensively studied.
- Silicosis—this occurs in miners who drill rock, sink shaft, or drive the transport cars ("motormen") and can be life threatening in severity.

There is a limited amount of work* that suggests increased respiratory disease in nonminer residents of coal mining communities, but this is not consistent between communities.

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*See Appendix K2 for references.

b. *Cardiovascular Disease Etiology*

Although there appears to be a moderate elevation in mortality in coal miners from "heart disease" in general and "myocardial infarction" in particular, these data must be interpreted very cautiously. An excess mortality from "heart disease" is a predictable result of a high prevalence of lung disease, because the pulmonary and cardiovascular systems are intimately related. Mechanisms for this interdependence are primarily the following:

(1) Progressive destruction of lung tissue also reduces the cross-sectional area of the pulmonary arterial bed. Reduced oxygen tensions also induce vascular constriction in the pulmonary artery. These phenomena result in reduced compliance, therefore increased resistance of the pulmonary arterial bed, which is normally a very low resistance hydrodynamic system. This increased resistance puts a markedly increased work load on the right heart; over time, the right ventricle hypertrophies (increases its muscle mass) to accommodate the need to generate excessive pressure. If the right ventricle cannot sustain the additional work, it will fail, a condition known as "right heart failure" which has several distinct features from the more common left heart failure. In addition, the hypertrophic right ventricle may demand more oxygen than its overextended coronary arteries are capable of supplying, producing angina pectoris. The phenomenon of right ventricular hypertrophy in association with lung disease is known as "cor pulmonale" and has been well documented to occur in coal miners with complicated coal workers' pneumoconiosis.

(2) Distension of the right atrium by the same mechanism described above and hypoxemia due to poor oxygen exchange by severe lung disease sensitize the heart to arrhythmias. In severe acid/base disturbances, which are not uncommon in chronic lung disease, the heart is also made more irritable. Foci in the cardiac conduction system and myocardium are thus more susceptible to spontaneous depolarization and may escape from electrical domination by the sinus node. Thus, irregularities in rhythm are among the most common consequences of moderate to severe lung disease. At the extreme, these arrhythmias can be fatal.

(3) Severe lung disease is a chronic process of symptomatic improvement followed by exacerbation in a stepwise downhill course. It is rarely a smooth history of decline, as cardiac disease more commonly may be. An intercurrent illness, such as influenza, may be all that is needed to acutely decompensate the patient and precipitate a cardiac problem. Unless a physician is attuned to this in his record-keeping, the interrelation may be lost in chart review.

(4) When a patient ceases to breathe as a result of lung disease and its attendant disturbance in gas exchange, the next event to occur (after brain damage, which does not declare itself immediately) is cardiac arrest. Undoubtedly, many deaths from lung disease are recorded as deaths from cardiac arrest.

The cardiovascular diseases which might be found in excess in coal mining communities would consist primarily of ICDA numbers 410 (myocardial infarction), 411 (acute/subacute ischemic heart disease), 412 (chronic ischemic heart disease), and 413 (angina pectoris). These are different manifestations of the same basic disease process: atherosclerotic coronary artery disease. If coal mining community residents are predisposed to accelerated atherosclerosis, these diseases should be uniformly elevated in risk.

c. Effects of Trace Metals

Certain minerals that can be solubilized by acid mine drainage (such as trace metals) have been linked to cardiovascular diseases by Schroeder [13]:

> "Cadmium is a causal factor in hypertension. A replica of the human disease was developed in rats and cured by removing the causal factor by chelation. Zinc deficiency could also play a part in hypertension."

Trace metals may be taken in, among other ways, through contaminated drinking water. For example, it is known that soft water, which is more corrosive than hard, can leach cadmium from water pipes and conduits.

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[13] Schroeder, H. A., The role of trace elements in cardiovascular disease, *Med. Clin. North Am.* *58*(2):381-396, 1974.

d. *Effects of Coal Combustion Products*

The adverse health effects of sulfur dioxide are well known and have been discussed in the copper smelting case study.

Nitrogen dioxide also exerts deleterious effects on the respiratory system. Animal experiments have shown that it provokes obstruction of the bronchi and rupture of the alveoli. For example, rats exposed intermittently for a lifetime (about 2.5 years) to 15 ppm of NO_2 developed emphysema, a chronic obstructive lung disease, and exhibited an attendant loss of oxygen-absorbing surface area in the lung. This resulted from destruction of tissue, with a loss of about two-thirds of the air sacs (alveoli). Stagnant air was retained in the enlarged, obstructed lungs following their removal from the chest. The main impact of injury initially was in the smaller airways that conduct gases immediately to and from the alveoli. These features constitute a reasonable facsimile of human emphysema [14].

Nitrogen dioxide likewise reduces resistance of the respiratory tract to infection. Prolonged exposure to considerable amounts of nitrogen oxides contained in tobacco smoke leads to chronic bronchitis. At the levels found in the atmosphere in certain cities, nitrogen dioxide is associated with an increase in acute respiratory problems among people of all ages. This is especially true of influenza epidemics. After two or three years of exposure to this risk, infections of the lower respiratory tract, bronchitis, and repeated respiratory infections become more frequent in children. The danger levels of nitrogen dioxide reported in winter are 160 $\mu g/m^3$ on the average with maximum levels exceeding 190 $\mu g/m^3$, 40 days out of 100 [15].

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[14] Workshop on "Possible Opportunity for Preventive and/or Therapeutic Medical Payoff from Grant-Supported Studies on O_3 and NO_2," sponsored by NCI, April 18, 1977.

[15] Gervois, M., and C. Dubois, Epidemiologic study of respiratory manifestations related to nitrogen oxides in urban air pollution, *Rev. Epidemiol. Med. Soc. 22*(8):710-713, 1974.

4. *Pathway Analysis*

The topography of Wyoming County is very distinctive. The entire county is crisscrossed by mountain ridges and narrow valleys, such that there is a total absence of open spaces or flatland. The ridges are steep and the valleys are narrow, giving them a canyon-like appearance. The sides of the "canyons" are roughly 1,800 feet high. Nearly all the population lives along the valley floors (in the hollows) which are never very wide—many times only a few hundred feet. Rivers and streams flow along most of the valley floors. The major coal preparation plants (thermal dryers) are located in the same valleys as the major population centers of Mullens, Pineville, Oceana, Kopperston, and Corrinne (see Figure VII-3).

The narrow valleys clearly define and channel the three main routes by which mine pollutants can reach the community, i.e., via water, air, and vehicles. Consequently, the three routes coincide, which makes it difficult to determine which route is responsible for an effect. In the case of the vehicular route, we clearly understand the pathway but there is no way to estimate concentrations or exposures to the populations at risk. For the water route, identifying the particular pathway by which the water from the mines or tipples reaches the population is difficult. However, we have been able to examine the quality of the water supplies in Wyoming County for evidence of industry-related pollutants, e.g., minerals solubilized by acid mine drainage.

a. *Air Quality Analysis*

In the case of the air route, no air quality measurements were available, so we cannot verify concentrations of either coal dust or coal combustion products. Taking current measurements would not solve the problem since the measurements would not represent conditions during the desired exposure period, i.e., the years prior to 1970 when emission controls were not in effect. In the absence of air quality data, some thought was given to point source dispersion modeling to estimate downwind concentrations. However, due to the peculiarities of the terrain and the lack of meteorological data for the region, it was felt that the modeling would not be meaningful. Consequently, we were not able to find or develop useful quantitative estimates of the amounts of toxicants arriving by the air route.

b. Water Quality Analysis

During site visits to West Virginia, sanitarians at the state and local levels provided water quality analyses of raw and treated municipal water supplies in Wyoming County. Most public water supplies in Wyoming County use well water. The averages of the annual analyses were compared against the Public Health Service (PHS) standards and the new EPA standards to determine if any violations of the standards existed which might pose an obvious threat to public health. Compiled data are presented in Table VII-6. Table VII-7 presents the standards against which the data were compared. Finally, Table VII-8 summarizes how the water supplies of Kopperston, Mullens, Oceana, Pineville, and Corrinne adhered to the standards shown in Table VII-7.

Judging from the high iron and manganese content of the Wyoming County municipal water supplies, it appears as if Wyoming County does suffer from some acid mine drainage (AMD) which has a tendency to solubilize iron and manganese, thus producing the high readings for those elements. High sulfate and total dissolved solids readings, also results of AMD, are not seen in the county, thus indicating that the problem is not as acute as in other coal mining regions.

Table VII-6. WATER QUALITY ANALYSIS: WYOMING COUNTY, WEST VIRGINIA

PARAMETER	AVERAGE FINISHED & RAW WATER QUALITIES, mg/ℓ (BY TOWNS)								
	Kopperston	Mullens		Oceana		Pineville		Corrinne	
	Finished	Finished	Raw	Finished	Raw	Finished	Raw	Finished	Raw
Alkalinity (phth)	0.0	0.0	0.0	1.5	0.0	0.0	0.0	0.0	0.0
Alkalinity (m.o.)	4.0	127.5	77.66	67.926	26.66	97.3	90.0	149.5	122.75
Hardness	22.0	131.5	82.0	104.5	48.0	64.0	57.3	76.0	134.0
Cadmium	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.03
Chromium	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Fluoride	0.12	0.33	0.15	0.56	0.12	0.40	0.15	0.47	0.085
Lead	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Silver	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chloride	7.0	19.25	19.0	34.0	9.0	5.0	18.6	18.05	7.75
Copper	0.006	0.025	0.0	0.0	0.10	0.01	0.0	0.0	0.27
Iron	0.09	0.52	6.51	0.39	3.84	0.19	18.57	2.93	19.8
Manganese	0.007	0.02	0.27	0.05	0.123	0.0026	0.58	0.46	0.5
Nitrate (as NO_3)	4.0	2.3	0.87	1.3	2.5	1.6	1.5	2.0	1.78
Sulfate	6.0	71.25	71.33	-	66.0	39.5	46.25	12.0	72.75
TDS	43.5	380.6	304.5	304.5	130.5	261.0	230.55	348.0	435.0
Zinc	0.05	0.20	0.037	0.08	0.05	0.12	0.13	0.05	8.54
Calcium	2.23	23.52	18.27	21.50	10.44	11.0	25.7	8.75	21.34
Magnesium	1.98	12.62	1.87	4.61	9.60	4.6	2.78	4.14	16.76
Sodium	0.98	41.63	52.4	8.5	31.57	39.1	51.0	55.37	91.17
pH (s.u.)	5.8	7.45	6.8	7.9	6.9	7.2	6.58	7.1	7.65
Turbidity (s.u.)	-	2.24	-	3.5	-	2.8	-	5.75	0.9
Potassium	0.96	4.23	7.17	1.58	2.00	-	4.42	1.07	1.74
Arsenic	-	0.005	0.005	-	-	0.005	0.0055	-	0.019
Nitrate (as N)	-	0.045	0.19	-	-	-	0.34	-	0.14
Mercury	-	0.00001	-	-	-	-	-	-	-

Table VII-7. DRINKING WATER QUALITY STANDARDS

Substance	USPHS (1962)	USEPA (1977)
Mineral Salts	(mg/ℓ)	(mg/ℓ)
TDS	500	-
Hardness	*	*
Calcium	-	-
Magnesium	-	-
Sodium	-	-
Alkalinity (phenolphthalein)	-	-
Alkalinity (methyl orange)	-	-
Chlorides	250	250
Fluorides	0.9-1.7	1.4-2.4
Nitrates	45	10
Sulfates	250	250
Trace Metals		
Arsenic	0.01	0.05
Cadmium	0.01	0.01
Chromium	0.05	0.05
Copper	1.0	1.0
Iron	0.3	0.3
Lead	0.05	0.05
Manganese	0.05	0.05
Mercury	-	0.002
Selenium	0.01	0.01
Silver	0.05	0.05
Zinc	5.0	5.0

*There is no standard for hardness. The following Classification of Water by Hardness Content Chart is provided by the EPA in *Quality Criteria for Water* (EPA 440/19-76-023):

Concentration mg/ℓ $CaCO_3$	Description
0-75	soft
75-150	moderately hard
150-300	hard
300+	very hard

Table VII-8

RESULTS OF COMPARISON OF AVERAGE CHEMICAL WATER QUALITY OF WYOMING COUNTY WATER SUPPLIES WITH PHS AND EPA STANDARDS[a]

PARAMETER	TOWN[b]	TYPE OF WATER	COMMENT
Cadmium	C	Raw	Exceeds all standards
Chromium	C,M,P,O,K	Raw/Finished	All in compliance
Fluoride	C,M,P,O,K	Raw/Finished	All in compliance
Lead	C,M,P,O,K	Raw/Finished	All in compliance
Silver	C,M,P,O,K	Raw/Finished	All in compliance
Chloride	C,M,P,O,K	Raw/Finished	All in compliance
Copper	C,M,P,O,K	Raw/Finished	All in compliance
Iron	P C,M,O	Raw Raw/Finished	Exceeds all standards
Manganese	M,P, O,C	Raw Raw/Finished	Exceeds all standards
Nitrate	C,M,P,O,K	Raw/Finished	All in compliance
Sulfate	C,M,P,O,K	Raw/Finished	All in compliance
Total Dissolved Solids	C,M,P,O,K	Raw/Finished	All in compliance
Mercury	C,M,P,O,K	Raw/Finished	All in compliance
Arsenic	C	Raw	Exceeds PHS standards
Zinc	C	Raw	Exceeds all standards

[a] If supply is not mentioned, it is assumed to be in compliance; no raw water was analyzed for Kopperston.

[b] Town code: C = Corrinne; M = Mullens; P = Pineville; O = Oceana;
K = Kopperston.

5. *Candidate Hypotheses*

Based on the identified pollutants, the etiology of respiratory and cardiovascular diseases, and the pathway analysis, candidate causal hypotheses were constructed. These were then examined in light of the evidence already developed to assess the plausibility of the hypotheses. The candidates and the results of the preliminary evaluation are presented in Table VII-9.

The etiology connecting coal dust and particulate matter to respiratory disease is well established, and there appears to be a plausible connection between cardiovascular disease and chronic respiratory disease. The population in Wyoming County is certainly exposed to coal dust from the tipples and fugitive vehicular emissions; however, in the case of mine emissions, the population is generally remote to the mine ventilation shafts. The principal difficulty with any of the coal dust hypotheses is the lack of reliable evidence of any kind on the magnitude of exposure. Observations on site visits disclosed that the communities are uncommonly dusty, even in this day when considerable care is taken to control the dust and particulate emissions from thermal dryers and vehicles. The narrow valleys help contain the dust to the populated regions, and the wind and vehicular traffic constantly stirring up accumulations of the dust in the valleys adds to the ambient coal dust concentrations. At this point, for purposes of hypothesis development, we will assume that ambient dust concentrations in the communities have been sufficiently high in the past to be instrumental in causing chronic respiratory and heart diseases. Since the thermal dryers are a major source of coal dust, it is reasonable to conjecture that populations living close to thermal dryers will be exposed to higher dust concentrations than remote populations. Hence, we would expect to find more prevalence of disease in the vicinity of thermal dryers than elsewhere. Thus, to explore the plausibility of the coal dust hypothesis, we will examine respiratory and cardiovascular disease patterns as described in the next section.

Table VII-9. *PRELIMINARY EVALUATION OF CANDIDATE HYPOTHESES*

HYPOTHESIS		EVALUATION CRITERIA			PLAUSIBILITY OF HYPOTHESIS
Etiologic Agent	Source	Plausible Etiology	Suitable Population At Risk	Sufficient Concentration	
Coal Dust & Particulates	Mines (Ventilation Shafts)	Yes	No	Unknown	Weak
	Tipples (Principally Thermal Dryers)	Yes	Yes	Unknown	Possible
	Vehicles	Yes	Yes	Unknown	Possible
Coal Combustion Products	Gob Piles	Yes	Yes	No	Weak
	Tipples (Thermal Dryers)	Yes	Yes	No	Weak
Acid Water (Solubilized Minerals)	Mines (Drainage)	Partly (Cardiovascular Dis.)	Possibly	No (Water Quality Analysis)	Weak
	Tipples (Coal Washing)	Partly (Cardiovascular Dis.)	Possibly	No (Water Quality Analysis)	Weak
	Gob Piles (Leachate)	Partly (Cardiovascular Dis.)	Possibly	No (Water Quality Analysis)	Weak

In the case of coal combustion products—in particular, the gaseous products from gob piles and thermal dryers—the emissions appear to be too low to provide a reasonable possibility of harmful effects in the community. Hence, these hypotheses are not considered to be plausible.

Likewise, the hypotheses based on a water route are considered to be too weak to be worthy of further consideration. The water quality analysis of public water supplies disclosed no hazardous levels of toxic agents. Although there is some evidence of acid mine drainage in the water system (e.g., elevated levels of manganese and iron), it is clear that this is not a major problem in the area due to low concentrations of sulfates.

Two additional hypotheses have been posed which are not directly related to the coal mining industry but which have an indirect connection. The first of these is the hypothesis that coal burning in the home is the source of excessive respiratory and cardiovascular diseases in the community. This hypothesis was not supported by the 1970 census information which disclosed that better than 90% of the households in Wyoming County use gas and oil for home heating. The second indirect hypothesis is that coal miners and their families are heavy smokers. This was verified by a local tobacco distributor and by hospital officials who felt that more than 90% of the miners smoked and most of their wives did likewise. In general, the miners chew tobacco when in the mines, where smoking is not allowed, and smoke cigarettes when out of the mine. Some further consideration is given to this hypothesis in the remainder of the case study.

6. *Mortality Analysis*

On the basis of the above analysis, the most plausible hypothesis for a causal relationship between industry and disease is the hypothesis that coal dust emissions from the preparation plants (principally thermal dryers) and other sources (coal transport vehicles and mine ventilation systems) are instrumental in causing respiratory/cardiovascular disease. To examine this hypothesis in light of mortality patterns, two separate investigations were made. In the first case, it was assumed that the thermal dryer is the major source of the coal dust and that communities close to the source would be exposed to a greater extent than distant communities. The major population centers in Wyoming County, which were all demographically very similar, were divided into two groups (those close to thermal dryers and those far from thermal dryers) and the mortality rates were compared.

In the second mortality investigation, it was assumed that the coal dust was an area problem associated with and accentuated by the particular mountainous terrain found in much of Appalachia. That is, it was assumed that coal operations in the narrow valleys create coal dust that is trapped in the valleys and pervades the entire population living in the valleys. In this case, the mortality patterns of counties with and without significant coal mining operations were compared for both mountainous and flat regions.

The results of these two mortality investigations are discussed below.

a. Mortality Study for Thermal Dryer Hypothesis

The major population centers in Wyoming County and the distance from the nearest thermal dryer are listed below:

Town	Population	Distance from Nearest Thermal Dryer (miles)
Kopperston	906	1
Mullens	2,985	3
Pineville	1,135	6
Oceana (Hatcher)	1,642	6
Corrinne	948	6

Because of their proximity to thermal dryers, the first two were considered to be the "exposed" population, and the last three were grouped as the "control" population

Copies of death certificates for Wyoming County for the years 1968 through 1972 were obtained from the Director of Registration and Public

Health Statistics, Health Department, West Virginia, for respiratory and cardiovascular diseases (see Table VII-1). The distribution of deaths from these diseases between exposed and control towns is given below:

Area	Town	Number of Deaths
Exposed	Mullens	32
	Kopperston	2
Control	Pineville	9
	Oceana (Hatcher)	26
	Corrinne	7

Because of the small number of deaths, we decided to treat the two diseases together rather than look at them separately. It is to be noted that we ordered death certificates for additional years and diseases, however this order was never filled due, presumably, to work overload of the Registrar and his staff.

To create the age-adjusted mortality rates, 1970 census population figures were used. Wyoming County is divided into 29 enumeration districts (EDs) of which the following districts match town boundaries:

Town	ED
Kopperston	1
Mullens	18,19,20,26
Corrinne	17,25
Pineville	13
Oceana (Hatcher)	2

The remaining EDs are rural and their boundaries follow streams or mountain ridges. As a result, small towns such as Herndon, Rock View, and Baileyville can be divided by two or more EDs. Also, the rural EDs are so large that they include both exposed and unexposed populations.

Using the 1970 census information, the demographic characteristics of the exposed study population were compared to those of the control population. Table VII-10 presents this comparison. It is evident that most of the socioeconomic factors considered are quite similar for the two groups. The significant differences occur in "% finished high school" and "% using coal for heating." In both these cases, the values for the control group are biased towards higher disease incidence, thereby increasing the suitability of the population for control purposes.

Table VII-10. SUMMARY OF DEMOGRAPHIC CHARACTERISTICS OF ENUMERATION DISTRICTS IN WYOMING COUNTY, WEST VIRGINIA

CHARACTERISTICS	EXPOSED	CONTROL
Median Male Age Group	25-34	25-34
Median Female Age Group	25-34	25-34
% Female	52	53
% Native of Native Parents	97	98
% White	95	96
% < 1.00 Persons per Room	95	87
% Electric Home Heat	1.8	3.7
% Coal Home Heat	1.5	10
% High School Grad	52	40
% At Same Residence as 1965	66	53
Median Income, $ × 1,000	8-9	7-8

To compare the respiratory/cardiovascular disease prevalence in the exposed and control areas, age-adjusted mortality rates were computed for white females (95% of the population is white). Since females do not work in mines, any differences in mortality rates for females should indicate a community health problem rather than an occupational problem. The significance in differences between exposed and control areas was tested using the Cochran test. The results are presented in Table VII-11.

*Table VII-11. MORTALITY STUDY RESULTS: WYOMING COUNTY**

	EXPOSED	CONTROL
Population	1,867	1,845
Mortalities	19	15
Age-Adjusted Mortality Rate/10^5	584	381
p (Cochran test)	0.04	

*Based on mortalities in white females, ages 0-74, during 1968-1972, from respiratory and cardiovascular diseases.

Although the number of deaths is small, the difference between the age-adjusted rates for the two areas is statistically significant. The probability is less than 0.04 that the two values would occur in the same population. This result provides some evidence in support of the thermal dryer coal dust hypothesis.

It is to be noted that this result tends to work against the two socioeconomic hypotheses, in which smoking and home heating by coal are assumed to be the cause of disease in coal mining communities. In the case of smoking, there are no apparent reasons why the exposed area should be any different from the control, since they are both located in the center of coal mining operations and occupationally and socioeconomically they should be very similar. So a difference in mortality rates cannot be readily blamed on tobacco usage.

In the case of use of coal for home heating, 10% of households in the control population used coal as opposed to 1.5% of the exposed. Hence, if home use of coal is the cause, then the control area should exhibit higher mortality rates than the so-called exposed area, which wasn't the case.

b. County Mortality Pattern Analysis

To shed light on the factors that might be important in the association between bituminous coal mining and respiratory/cardiovascular disease, we performed a regional analysis of the SSI county mortality data. For the mountainous regions of West Virginia, Kentucky, and Tennessee, we divided all the counties into three categories with respect to the amount of coal mining in the county: high production, low production, and no production. For each state, we computed the average county mortality rates in each category for two disease groups: respiratory disease and cardiovascular disease. We then did the same thing for the flat regions of Ohio, Illinois, and Kentucky. In both cases, we eliminated all counties with cities and all counties with populations less than 5,000, so that all counties used had populations in the range of 5,000 to 27,000 for the 35-74 age bracket. The results are presented in Tables VII-12 and VII-13.

Table VII-12

EFFECT OF COAL MINING ON COUNTY MORTALITY RATES IN MOUNTAINOUS TERRAIN (WEST VIRGINIA, TENNESSEE, KENTUCKY)

State	Coal Production	No. of Counties	AGE-ADJUSTED MORTALITY RATES in White Females, ages 35-74	
			Respiratory Disease	Cardiovascular Disease
West Virginia	None	12	39	451
	Low	10	51	485
	High	8	51	519
Tennessee	None	18	48	403
	Low	5	46	414
	High	0	--	--
Kentucky	None	2	33	440
	Low	4	38	438
	High	5	40	515
Total for Three States	None	32	44	423
	Low	19	47	456
	High	13	47	517
National Average:	All U.S. Counties		37.5	397

For mountainous terrain, these data show a clear trend between the amount of industrial activity and the average county mortality rates for cardiovascular diseases. This trend is pronounced in each state and in the aggregation of all states.

In flat terrain, we see a similar trend, but in this case it is much less pronounced. For example, the relative risk between high-production and no-production counties is 1.24 for mountainous terrain and only 1.07 for flat terrain. The difference between these relatives rates is quite significant considering the number of counties involved and the regional similarities between the "exposed" and control counties. The fact that for counties with no coal production the aggregated mortality rates for the two types of terrain are fairly close adds to the significance of the difference in rate ratios between mountainous and flat terrains.

Table VII-13

EFFECT OF COAL MINING ON COUNTY MORTALITY RATES IN FLAT TERRAIN (KENTUCKY, OHIO, ILLINOIS

State	Coal Production	No. of Counties	AGE-ADJUSTED MORTALITY RATES in White Females, ages 35-74	
			Respiratory Disease	Cardiovascular Disease
Kentucky	None	7	34	363
	Low	0	--	--
	High	2	28	407
Ohio	None	7	43	430
	Low	2	41	459
	High	1	56	466
Illinois	None	5	40	469
	Low	4	38	446
	High	1	23	489
Total for Three States	None	19	39	416
	Low	6	39	450
	High	4	34	442
National Average:	All U.S. Counties		37.5	397

A possible inference from these data is that the type of terrain is a factor in the association between industry and cardiovascular disease. If we put this finding together with our coal dust hypothesis, a plausible explanation is that the valleys in mountainous terrain tend to confine the coal dust to the communities; whereas in flat terrain, the coal dust is more widely dispersed.

Looking at respiratory diseases, we see that the mortality rates tend to be correlated with coal production in mountainous terrain, but the trend is not as strong as in cardiovascular disease. Furthermore, in flat terrain there is no clear association between respiratory disease and coal mining production. Although this can be interpreted as refuting the coal dust hypothesis, it can be explained as being compatible with the hypothesis. If we assume that chronic respiratory disease can lead to death from cardiovascular stress prior to death from respiratory disease, then this can explain why the increase in cardiovascular disease is more pronounced. This is a reasonable explanation which is hard to prove without examination of autopsy records to see if there was a higher incidence of cardiovascular decedents with respiratory problems in coal mining regions.

The results of this regional county analysis tend to negate the smoking hypothesis, i.e., the hypothesis that coal mining operations encourage heavy use of tobacco which extends to families and communities. If smoking were the cause, there should be no difference in relative rates between mountainous and flat terrains.

7. *Discussion of Hypothesis Development Results*

Although the evidence is not strong, it appears to point to the hypothesis that coal dust from coal mining operations is the source of chronic respiratory and cardiovascular diseases in coal mining communities. A major source of the coal dust is the coal preparation plants, particularly the large thermal dryers which are the focal points of coal handling and transport operations and are located in the same valleys as the major population centers. This hypothesis is supported by the analysis of respiratory and cardiovascular mortalities in the county, which shows that the mortality rates in females are higher in the vicinity of thermal dryers than elsewhere. A contributing factor may be the canyon-like valleys which confine the coal dust to populated areas. The analysis of regional county mortality patterns shows that the relative mortality rates between coal mining and non-coal mining counties are higher for mountainous terrain than for flat terrain. One of the strongest arguments in favor of the coal dust hypothesis is the etiology of respiratory disease in coal miners, which establishes a definite causal connection between coal dust and respiratory disease.

The principal weakness in the coal dust hypothesis is the lack of ambient air quality data to show that the communities are indeed exposed to hazardous levels of respirable size coal dust. In present-day operations, the thermal dryers use highly efficient control devices which collect as much as 99% of the coal dust and particulate emissions. No measurements are available to show the ambient conditions prior to 1969 when the present-day control devices were installed.

Other possible hypotheses appear to lack one or more key elements. The combustion products from thermal dryers and other sources are not produced in sufficient quantities to be hazardous. Source strengths are much less than found in metropolitan areas throughout the county. Water quality is generally good in Wyoming County, and there are no hazardous levels of harmful substances that are by-products of a severe acid mine drainage problem. Furthermore, water pollution from mine operations cannot be connected with respiratory diseases.

Home heating with coal does not appear to be the source of the excess diseases, because a very high percentage of the households use gas or oil heating and the mortality analysis shows a lower mortality rate in the area with highest usage of coal for home heating. However, we may be seeing the latent health effects of the exposure that occurred years ago when coal for home heating was more popular. So this hypothesis cannot be completely disregarded. Neither can we convincingly disregard the smoking hypothesis. Although the mortality patterns tend to work against this hypothesis, there is insufficient data to make a strong case either way.

There is always the possibility that migration can cause the mortality patterns, particularly at the county level. In coal mining areas in general and in Wyoming County in particular, the populations have been fairly stable. The general trend during the 1950's and 1960's has been an emigration of young people, such that most coal mining areas show a negative net migration during the period of most interest; in Wyoming County, the net migration between 1960 and 1970 was -24.4%. Even if we assume that enough young people move out such that a severe change in age distribution occurs, the age-adjusted mortality rate would not change. The type of migration that affects epidemiological analysis is a selective migration of sick or healthy people. To effect an increase in mortality with negative migration, the emigrants must be more healthy than those remaining. There is no evidence that this has been the case for coal mining regions.

C. HYPOTHESIS TEST

1. Objective

To test the coal dust hypothesis, two independent investigations were carried out. The first was the collection and analysis of hospital patient records in Wyoming County, West Virginia, to determine morbidity patterns. The second was a field investigation (primarily a mortality pattern study) at a new site. For this site, a five-county area in southern Illinois was selected. Besides being a heavy coal producer with a number of large thermal dryers, the area appeared to have good population-at-risk characteristics. Furthermore, mortality data were readily accessible, which had not been the case in Wyoming County. The results of these two hypothesis test investigations are discussed below.

2. Wyoming County Morbidity Study

a. Approach

There are six hospitals in the vicinity of Wyoming County; however, patient flow information supplied by the West Virginia State Department of Health showed that three hospitals admit the large majority of Wyoming County patients. The hospitals and the estimated percent of study site total admissions served by the hospitals are listed below.

Hospital	Town	Percent of Total Study Site Patients
Stevens Clinic	Welch	23
Appalachian Regional	Beckley	20
Wyoming General	Mullens	32

On the basis of this information, it was decided to analyze morbidity records for Wyoming County patients from the three principal hospitals serving Wyoming County. Although all three hospitals initially voiced support for our study and gave tentative approval to supply us with patient data or records, only one (Wyoming General Hospital) could obtain official approval and could provide the patient data on a time scale and in a manner compatible with our study requirements. Although this severely reduced the effectiveness of the morbidity analysis because of the unknown and indeterminable hospital-selection biases of the patients, we decided to proceed with the Wyoming General Hospital data since the hospital treats a significant percentage of the Wyoming County patients and, further, we felt we had ways to analyze the data that would eliminate much of the selection bias.

Due to the expense of data collection and to the fact that we were primarily interested in environmental as opposed to occupational diseases, we collected hospital records for females only. We felt that the disease stage might provide valuable information for assessing toxic effects, hence staging was performed on 50% of the records collected.

The objective of the morbidity data analysis was to identify disease residence patterns, e.g., to determine if disease incidence or severity is a function of the distance of residence from a thermal dryer. To accomplish this, the prevalence of the diseases of interest (respiratory and cardiovascular) was determined as a function of town of residence. However, to reduce the effect of selectivity bias, we used a measure of prevalence consisting of the ratio of the disease-specific cases to the total number of cases from all diseases less the disease-specific cases. If, in a given community, the probability that a patient will select Wyoming General Hospital is the same for respiratory and cardiovascular diseases as it is for other diseases, then the ratio gives an unbiased measure of the relative prevalence between communities. However, if there is appreciable disease selectivity and the patient selects different hospitals for different diseases, then the results will be biased and less valid. We do not know at this point the magnitude of disease selectivity; hence, the results must be interpreted in the light of this unknown.

The data abstracting included histories and other lifestyle information; however, too little of the data was available on the records to permit a meaningful analysis.

b. Morbidity Data Collection

The morbidity data collection at Wyoming General Hospital in Mullens, West Virginia, was carried out using seven local individuals who performed abstracting work and clerical support such as pulling and refiling charts.

Medical records were abstracted for white females in the towns of Corrinne, Kopperston, Mullens, Oceana (Hatcher), and Pineville. Records date back to the late 1950's. All diseases (except respiratory and cardiovascular) were abstracted on the short form. Respiratory and cardiovascular diseases were abstracted on the long form. (See Appendix L which presents the Hospital Record Abstract Forms.)

The entire hospital file was screened in order to identify the patients of interest. After 50% of the records had been abstracted, the use of the long form was discontinued and the short form was used for respiratory and cardiovascular patients. This was done in the interest of time and expense, and because it was believed that a 50% sample using the long form would be adequate to assess the usefulness of the staging data.

The following conditions were not abstracted: routine surgeries (such as tonsils and hemorrhoids), obstetrical cases, accidents, tooth extractions, and fractures. Although these conditions were not abstracted, their numbers were recorded.

The procedure employed involved: (1) identifying the charts of interest, (2) pulling and screening of charts to eliminate routine surgeries, obstetrical cases, etc., (3) abstracting, and (4) refiling the charts.

In step 1, the clerk manually screened the chart number index cards which were alphabetical by last name and included the address, race, chart number, dates of admissions, in- or out-patient status, and active or inactive status. The sex was determined by the first name, and the chart was pulled when the sex was questionable which was rather frequent in that area. The chart number index consisted of nine drawers of 3-by-5 file cards; each drawer had three columns. The first column in all of the drawers was screened first, then the second and third columns. This "random" sampling method was used in case the study had to be stopped short of completion due to time and/or funds limitations.

As the chart numbers were identified, they were listed on the coding forms (see Appendix L). An "I" was added to the chart number for the inactive patients (those not hospitalized in the last five years) because they were stored separately from the active files.

The abstractors located and pulled each identified chart, then quickly scanned it. Charts with nondisease conditions (accidents, etc.) were refiled immediately and a notation made beside the code number. The remaining charts of interest were collected and abstracted.

Respiratory and cardiovascular diseases were abstracted using the long form. For repeated diagnoses, staging forms were used only for the first three admissions. All other diseases were abstracted using the short forms (see Appendix L). When a file required a long form but not all admissions were respiratory or cardiovascular, only the staging forms were used. After abstracting the records were refiled.

An evaluation of the data collection was made on the third day. Approximately one full day of abstracting had been completed by two abstractors. More than 50% of all cases were out-patients, and their files were not coded with an HICDA number, were incomplete and unsummarized; as a result, these were omitted from the study. About 50% of the remaining cases had minor surgery, fractures, accidents, etc., and created record-handling problems greater than their value; consequently, their numbers were recorded (during the record scans) but they were not abstracted. In the first full day of abstracting, about 40 records were processed—one-third of which required the long form.

The short forms required about two to three minutes, and the long forms about 5 to 20 minutes.

The following listing summarizes the data collected:

• Charts pulled	2,603
• Charts of miscellaneous diseases	379
• Charts of respiratory diseases	97
• Charts of cardiovascular diseases	108
• Total charts abstracted	584

c. *Results of Morbidity Analysis*

After the elimination of a small number of unusable records, a total of 565 medical record histories remained which represented 565 separate individuals. These had been coded into respiratory and cardiovascular disease categories by the abstractors according to the category definitions shown in Table VII-1. Table VII-14 gives the number of admissions by major disease category and town.

Table VII-14

NUMBER OF HOSPITAL ADMISSIONS BY DISEASE CATEGORY AND TOWN

Town	Respiratory Diseases	Cardiovascular Diseases	Other Diseases	Total by Town
Kopperston	2	1	8	11
Corrinne	4	6	26	36
Mullens	54	54	190	298
Oceana	15	22	44	81
Pineville	20	24	95	139
Total by Disease Category	95	107	363	565

To test the thermal dryer hypothesis, the admissions were grouped in the hypothesized exposed and control areas where Kopperston and Mullens are considered to be the exposed towns. To eliminate the hospital selectivity bias, the "other diseases" were taken as control diseases which would exhibit the same selectivity as the respiratory and cardiovascular diseases. Therefore, the ratio of respiratory or cardiovascular diseases to other diseases would represent the relative disease prevalence which can be compared between exposed and control areas. Table VII-15 presents these results.

Table VII-15

COMPARISON OF RELATIVE DISEASE PREVALENCE BETWEEN EXPOSED AND CONTROL AREAS*

Town	DISEASES					
	RESPIRATORY		CARDIOVASCULAR		OTHER	
	Number of Admis.	Relative Disease Prevalence	Number of Admis.	Relative Disease Prevalence	Number of Admis.	Relative Disease Prevalence
Exposed[a]	56	0.28	55	0.28	198	1
Control[b]	39	0.24	52	0.32	165	1

*Relative Disease Prevalence = $\frac{\text{Number of Admissions}}{\text{Number of "Other Diseases" Admissions}}$

[a] Kopperston, Mullens [b] Corrinne, Oceana (Hatcher), Pineville

It is immediately apparent that there is no appreciable difference in relative disease prevalence between the postulated exposed and control regions, for either respiratory or cardiovascular diseases. Hence, these results are negative with regard to the thermal dryer hypothesis.

As a check of this result, this comparison was made by an independent method. In this case, the results of a patient flow study of Wyoming General Hospital admissions were used to estimate the expected distribution of diseases between exposed and control regions. On the basis of the results of two surveys of admission patterns, it was determined that out of 64 total admissions during the survey period, 60% were persons with residences in the exposed area. Thus, in order for the exposed area to indicate a higher prevalence of disease, the ratio of the number of admissions for the exposed to that for the control must be greater than 1.5 (60%/40%). Clearly this is not the case in Table VII-15; for respiratory diseases the ratio is 1.43 (56/39) and for cardiovascular diseases it is 1.05 (55/52).

Data from the National Health Survey [16] were used to estimate the fraction of hospital admissions with respiratory or cardiovascular disease. This was then compared with similar fractions for Wyoming County. Table VII-16 gives the results. These data show that the fraction of admissions is higher for Wyoming County. This could indicate that the prevalence of both respiratory and cardiovascular diseases is higher than normal.

Table VII-16

*COMPARISON OF WYOMING COUNTY HOSPITAL ADMISSION DATA TO THAT OF SOUTHERN UNITED STATES**

	Respiratory Diseases	Cardiovascular Diseases
Number of Admissions	95	107
Fraction of All Diseases: Wyoming County	0.17	0.19
Fraction of All Diseases: Southern United States[a]	0.12	0.11

**Source:* Reference [16].

[a] Based on a total of 7,295,000 hospital admissions in 1972.

To complete the exploration of the Wyoming County admission data, a detailed examination of the data was performed by disease stage, by specific ICDA number, and by age. The attempt was to ascertain if there were any patterns in the data that would be illuminating. This detailed examination did not reveal anything new and showed the general lack of significant differences between exposed and control areas for specific diseases, stages, and ages.

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[16] *In-Patient Utilization of Short-Stay Hospitals by Diagnosis - United States - 1972*, U.S. Department of Health, Education and Welfare, November 1975.

3. *Southern Illinois Field Study*

a. *General Site Characteristics*

The bituminous coal mining district of southern Illinois (Randolph, Perry, Franklin, Williamson, and Saline Counties) was selected for a test site. Reasons for the selection were the presence of extensive coal mining (both surface strip and underground), associated tipples, and the potential exposure of large populations (e.g., ∿13,000 in Harrisburg, Illinois, vs. ∿3,000 in Mullens, West Virginia). Also, mortality data by town of residence, cause of death, age, race, sex, and year of death were readily available from the Illinois Department of Public Health, State Center for Health Statistics.

The terrain consists of gently rolling hills, much less distinctive and confining than Wyoming County. Figure VII-6 is a map of the area showing the major towns and coal-mining operations.

There are 10 major tipples in the study area (see Table VII-17). Nine have operating histories dating back to the 1940's under various company names as the plants were bought and sold. Thermal dryers are operated by five of the tipples. These five are located on the map shown in Figure VII-6. Only three of the dryers are sufficiently close to towns to constitute a potential problem to the towns. Table VII-18 lists these three tipples and associated populations at risk (towns) and distance between the tipples and towns.

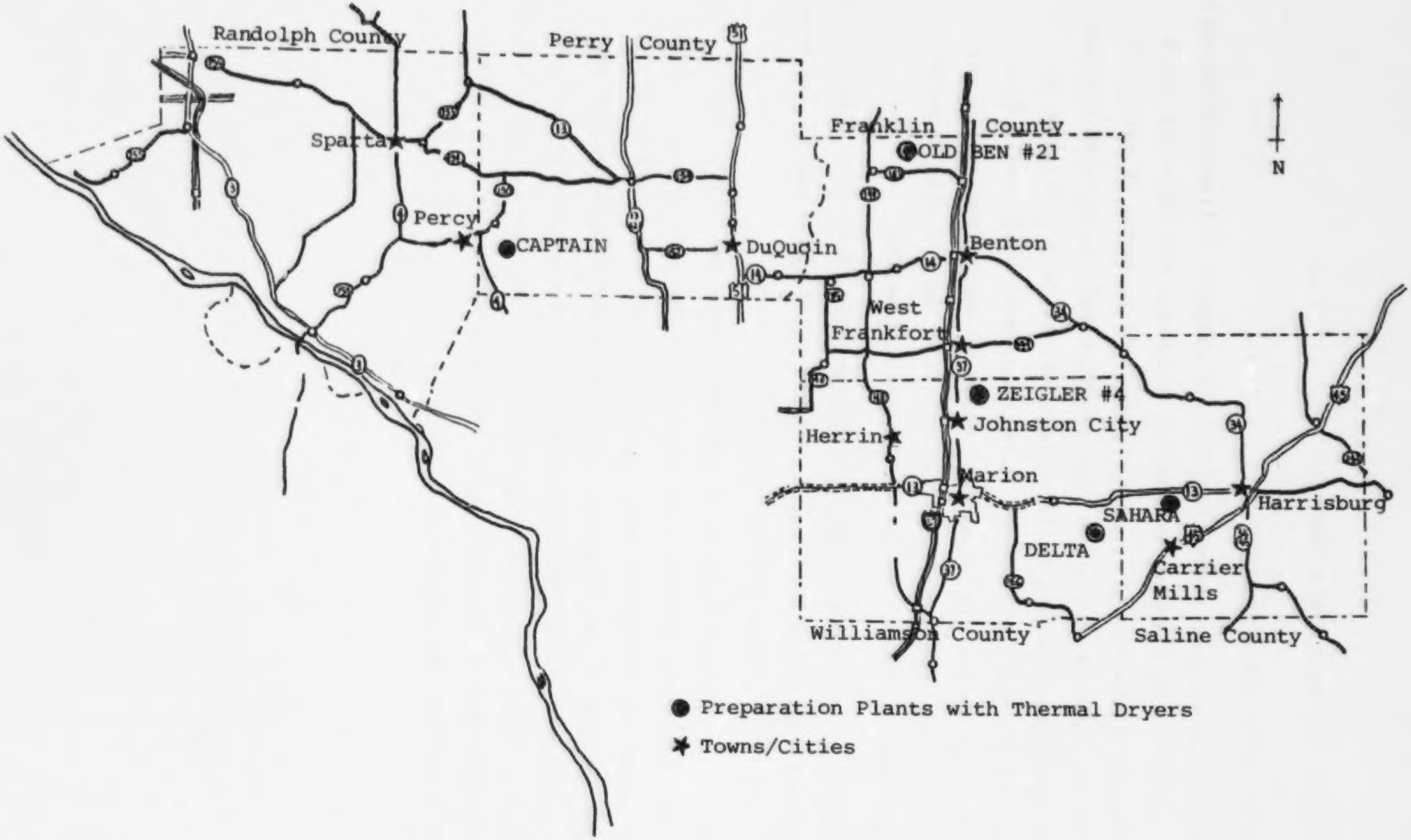

Figure VII-6. Map of Southern Illinois Study Area: Major Towns and Coal-Mining Operations

*Table VII-17. PREPARATION PLANTS: SOUTHERN ILLINOIS**

Preparation Plant (Company)	Thermal Dryer	Closest Town
Captain (Southwestern Illinois)	Yes	Percy
Streamline (Southwestern Illinois)	No	--
Orient #4 (Freeman)	No	--
Orient #5 (Freeman	No	--
Central (Sahara)	Yes	Harrisburg
Zeigler #4 (Bell & Zoller)	Yes	Johnston City
Delta (Ayshire)	Yes	--
Old Ben #21 (Old Ben)	Yes	--
Old Ben #26 (Old Ben)	No	--
Old Ben #27[a] (Old Ben)	--	--

* *Source:* Reference [4].

[a] New plant.

Table VII-18

RELATIVE LOCATION OF TOWNS FROM THERMAL DRYERS: SOUTHERN ILLINOIS

Tipple (Company)	Direction of/Distance to Town from Tipple Min & Max, miles	Town/Population
Central (Sahara)	E/2.3 - 4.6 S/3.0 - 3.6	Harrisburg/13,000 Carrier Mills/2,000
Captain (Southwestern Illinois)	WNW/6.0 - 6.5	Percy/1,000
Zeigler (Bell & Zoller)	WSW/1.7 - 3.1	Johnston City/4,000

b. Transport Analysis

No air quality information was available for the towns of interest in the southern Illinois study site. Consequently, calculations were made using a stack dispersion model to estimate a measure of coal dust exposure to the populations in the vicinity of the thermal dryers. It is felt that the results of this type of modeling have much greater applicability to the type of terrain in Illinois than to that in Wyoming County, West Virginia.

Turner's calculation method described in Equation (4) of Appendix A was used to estimate exposure of two southern Illinois communities to emissions from thermal dryers at coal preparation plants. These communities were Harrisburg, Illinois, 5 to 7 km due east of the Sahara mine and preparation plant, and Johnston City, 5 to 7 km west-southwest of the Zeigler mine and preparation plant. This report presents input parameters and results for the model, methods of constructing isopleths, applicability of results, and a short critique of the applicability of the Turner model to these sites.

Input data for the Turner equation were obtained from the Illinois EPA's Total Air System File (Springfield, Illinois) and from the National Climatic Center's STAR data files (Asheville, North Carolina). The point source data for the two sites and some meteorologic information are summarized in Table VII-19. Plume rise was calculated according to the method of Holland (see Appendix A). The STAR data used were obtained from observing stations in Evansville, Indiana, 80 miles east of the study sites. This STAR program was based on observations from January 1960 to December 1964, 24 observations per day. It was run for six Pasquill stability classes—three unstable, two stable, and one neutral.

Estimated values for long-term concentration of thermal dryer emissions (X) were calculated for hypothetical receptors at 0.25 to 10 km from each plant in 16 directions (points of the compass). Topographic maps of the study sites were obtained (U.S. Geological Survey, Reston, Virginia) and the calculated concentrations placed on a radial grid at appropriate distances and directions. In constructing concentration isopleths, high values to the northwest and southeast were smoothed out since they represented characteristics of Evansville meteorology rather than meteorology of the study areas. Although this determination alters the shape of the isopleth, it should not alter the meaning of the calculated values since stability categories in Evansville are similar to those in southern Illinois. Isopleths are drawn around the Sahara and Zeigler point sources in Figures VII-7 and VII-8, respectively.

Table VII-19

INPUT DATA FOR ESTIMATED LONG-TERM CONCENTRATIONS OF THERMAL DRYER EMISSIONS IN THE VICINITY OF HARRISBURG AND JOHNSTON CITY, ILLINOIS

Parameter (units)	Preparation Plant Data	
	Zeigler	Sahara
Inside stack diameter (m)	4.57	1.5
Physical stack height (m)	27.4	21
Atmospheric pressure (millibars)	1013	1013
Ambient temperature (°K)	286.8	286.8
Stack gas temperature (°K)	360.8	324.7
Stack gas exit velocity (m sec^{-1})	2.08	15.5
Wind speed (m sec^{-1})	1.5	1.5
Effective stack height (m)	52.88	51.55
Emission rate (g/sec)[a]	38.1	156.2

[a] Uncontrolled.

Applicability of the Turner analysis must be addressed. The only ambient air quality data for southern Illinois is from the towns of Effingham, Carbondale, Mt. Vernon, Marion, and Metropolis. The average of the annual geometric means for total suspended particulates (1976) in towns for which statistically significant data were reported (Effingham, Mt. Vernon, Marion) was 56 mg/m^3 [17]. Air quality of this magnitude is among the best in the state.

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[17] Illinois Environmental Protection Agency, Division of Air Pollution Control, *1976 Annual Air Quality Report*, Springfield, Illinois, 1977.

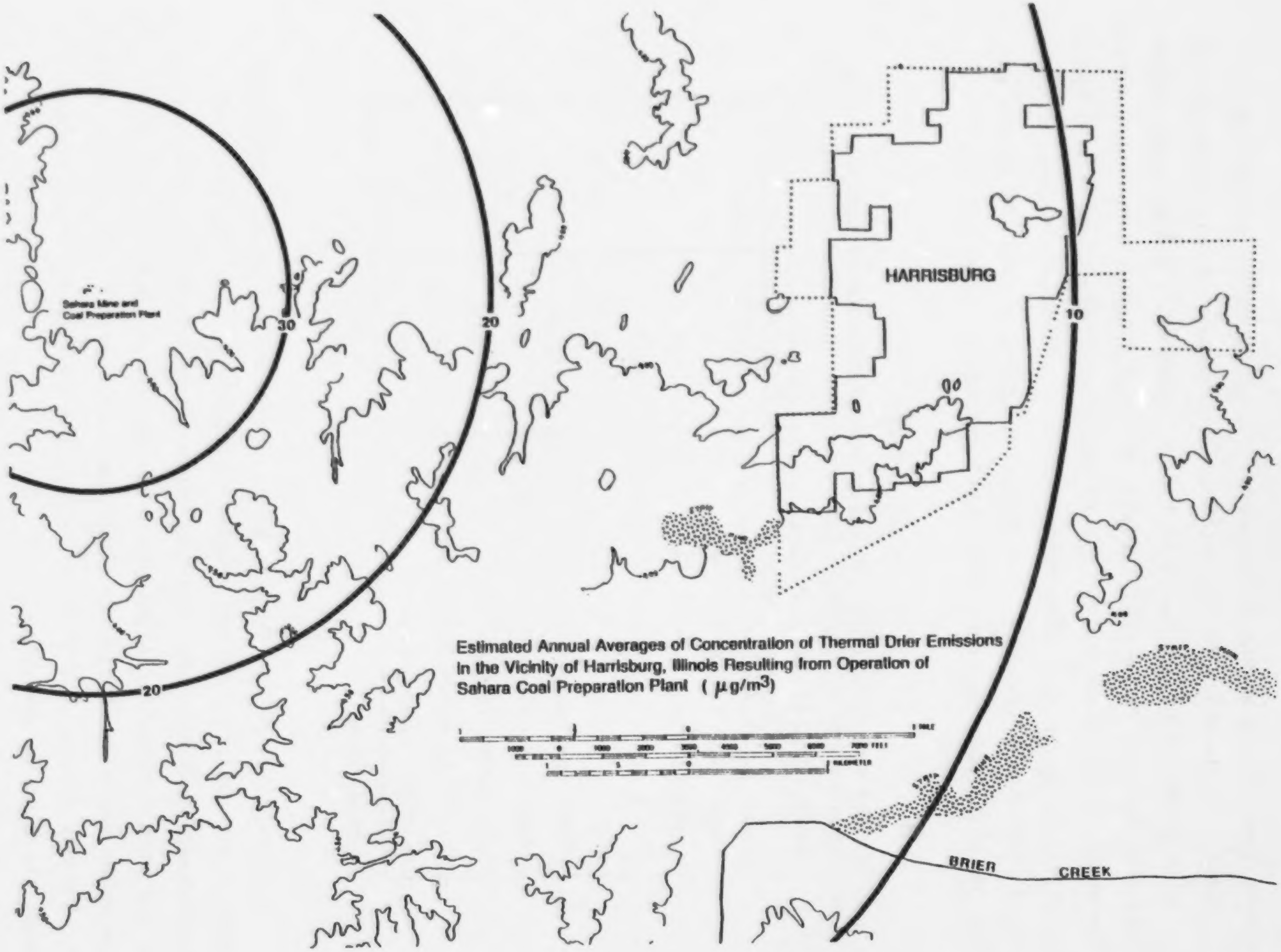
Sahara Mine and Coal Preparation Plant
30
20
20
10
HARRISBURG
Estimated Annual Averages of Concentration of Thermal Drier Emissions In the Vicinity of Harrisburg, Illinois Resulting from Operation of Sahara Coal Preparation Plant ($\mu g/m^3$)
BRIER
CREEK

Figure VII-7. Concentration Isopleths: Sahara Coal Preparation Plant and Harrisburg, Illinois

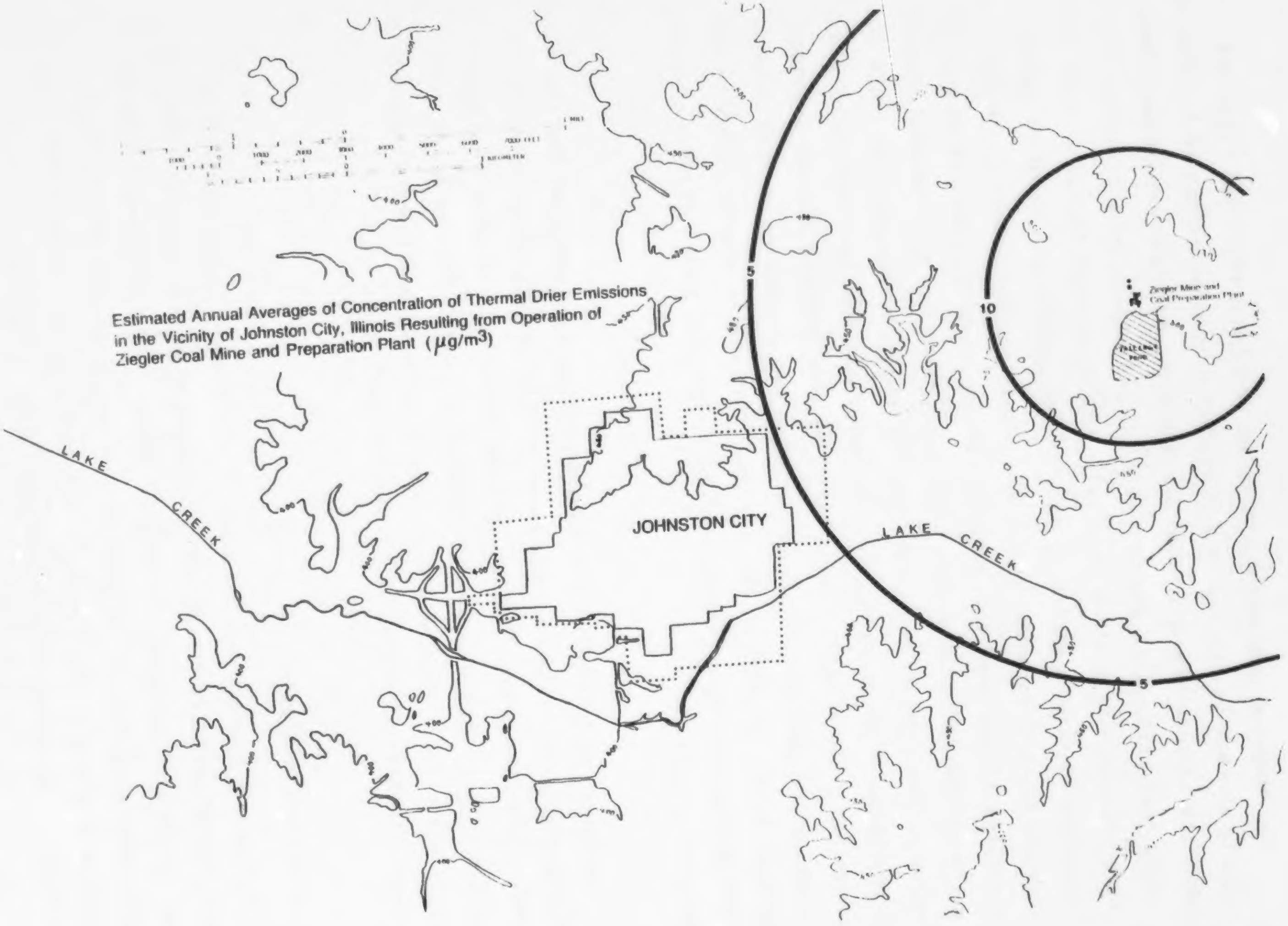
Estimated Annual Averages of Concentration of Thermal Drier Emissions in the Vicinity of Johnston City, Illinois Resulting from Operation of Ziegler Coal Mine and Preparation Plant ($\mu g/m^3$)
JOHNSTON CITY
LAKE CREEK
LAKE CREEK
5
10
5
Ziegler Mine and Coal Preparation Plant

Figure VII-8. Concentration Isopleths: Zeigler Coal Preparation Plant and Johnston City, Illinois

The computed concentrations shown in Figure VII-7 and VII-8 are yearly average values and do not represent concentrations to be expected when the tipples are operating and the meteorological conditions favor heavy town pollution. Further, the concentrations do not include fugitive coal dust from other sources in and around the tipple or from reentrainment of accumulations of dust in the community. Based on these considerations, we would expect the actual concentrations to be considerably higher than the computed values; however, there is no way to estimate how much higher they would be. As it is, the computed values shown in the figures are quite low compared to concentrations found in mines (tens of micrograms per cubic meter compared to hundreds of micrograms per cubic meter). Thus, the transport analysis has not provided verification that the population at risk was exposed to harmful levels of coal dust. At best we can say that the results are inconclusive and that air quality measurements must be found if a more conclusive answer is to be provided.

It is to be noted that the concentrations were computed for the case of uncontrolled emissions, which was largely the situation prior to 1969. In recent years, EPA has adopted emission standards for tipples and the mining companies have incorporated control devices which are highly efficient. Thus, we would expect a less significant relationship between tipples and community disease in today's environment.

c. Mortality Analysis

To further explore the validity of the thermal dryer hypothesis, mortality patterns at the southern Illinois site were studied. The towns in closest proximity to thermal dryers were considered to be the populations at risk, and mortality rates were compared between these towns and a set of control towns which were remote to coal preparation operations. The study and control towns are as follows:

Study Towns: Percy, Johnston City, Carriers Mills, Harrisburg

Control Towns: Marion, Herrin

Both the study and control populations are located in the same geographical area and have similar socioeconomic characteristics. Table VII-20 shows how closely the two areas are matched in race distribution and in number of persons per room, a key measure of lifestyle.

Table VII-20

STUDY AND CONTROL SOCIOECONOMIC INDICATORS: SOUTHERN ILLINOIS

Indicator	Study Area		Control Area	
	Number	%	Number	%
Race				
White	15,913	97	20,993	98
Negro	495	3	321	2
Other	34	0	33	0
Total	16,443	100	21,347	100
Persons per Room				
1.00 or less	140	64	208	64
1.01 to 1.50	14	6	13	4
1.51 or more	65	30	104	32

The numbers of mortalities, by year of death (1974-1976), cause of death, residence, age, race, and sex, were acquired from the Illinois Department of Public Health, State Center for Health Statistics. Age-adjusted mortality rates in females were computed for respiratory, cardiovascular, and "all other diseases," and differences in rates were tested for significance by the Cochran test.

The results, presented in Table VII-21, show that both respiratory and cardiovascular diseases are elevated in the population at risk, but only the cardiovascular difference is statistically significant. The mortality rates for all other diseases are similar for the study and the control populations. This provides evidence of disease specificity.

Table VII-21

MORTALITY STUDY RESULTS: SOUTHERN ILLINOIS

		Cause of Death		
		Respiratory Diseases	Cardiovascular Diseases	All Other Diseases
Age-Adjusted Mortality Rate/10^5 White Females	Study	94	479	611
	Control	62	359	691
Relative Risk		1.5	1.3	0.9
p (Cochran test)		0.21	0.01	--

4. Discussion of Hypothesis Test Results

Of the two tests performed on the coal dust hypothesis, the result of one was negative and the other, positive. The morbidity study in Wyoming County provided the negative result, which was the lack of a disease pattern associated with the thermal dryers. The mortality study in southern Illinois provided the positive result, which was the elevation of respiratory and cardiovascular disease in the vicinity of thermal dryers.

The morbidity result is not considered particularly significant because of the hospital selectivity problem. Due to the limited sampling of the hospitals in the area, we had no way of compensating for disease-specific differences in patient flow from the study and control areas. Probably the only thing that can be concluded from the morbidity analysis is that there is not a very large difference in respiratory and cardiovascular disease prevalence between the exposed and control areas. That is, if a relative risk does exist between the two areas, it is not outstanding.

The southern Illinois investigation provided significant evidence that females living near thermal dryers have a higher risk of death from cardiovascular disease than those living elsewhere. They also seem to have a higher risk of respiratory disease; however, the evidence is not quite as strong. The problem with this result is that we cannot produce concrete evidence that connects the excess mortalities to the thermal dryer. Air quality measurements are not available, and modeling of the coal dust dispersion indicates that the coal dust concentrations in the communities at risk might be too low to be harmful. Although we believe that the socioeconomic characteristics of the control and "exposed" areas are very similar, there is always the chance that the differences in the mortalities are caused by a difference in lifestyle.

At this point it is fair to state that the results of the hypothesis test effort are inconclusive. Examining all the evidence, pro and con, that has been developed to date, the balance sheet looks like this with regard to the coal dust hypothesis:

Pro

- Counties with bituminous coal mining have higher mortality rates in respiratory and cardiovascular diseases than do counties with no coal mining (both the regression analysis and the regional county pattern analysis give strong confirmation of this).
- In Wyoming County and in southern Illinois, communities close to thermal dryers have higher mortality rates in respiratory and cardiovascular diseases than do distant communities (these are statistically significant results except for respiratory diseases in southern Illinois).
- Coal dust is a known etiologic agent for respiratory disease and its connection to cardiovascular disease is medically plausible.
- The relationship between county mortality rates and coal mining activity appears to be stronger in mountainous regions than in flat regions. (This provides indirect support to the coal dust hypothesis; i.e., the explanation is that the coal dust concentrations are enhanced by the confinement of the valleys and lessened by the dispersal over flat land.)

- The percentage of hospital patients with respiratory and cardiovascular diseases in the Wyoming County study site is higher than average for Southern United States.
- No other reasonable explanation for the correlation between coal mining and respiratory/cardiovascular disease has been found which is supported by the evidence at hand.

Con

- The exposure of coal mining communities to high levels of respirable size coal dust cannot be verified. Measurements are unavailable and dispersion model computations do not confirm the existence of significantly high levels of dust concentration in the population at risk.
- Morbidity patterns in Wyoming County do not confirm that disease prevalence is higher in the vicinity of thermal dryers (this negative result is not particularly significant due to insufficient data to derive conclusive results).
- Duration of exposure (duration of residence) of decedents in the exposed population was not confirmed.
- Other socioeconomic or environmental causes of excessive mortality cannot be ruled out, such as excessive use of tobacco or home heating with coal. There is some evidence against these causes, but it is not sufficiently strong to preclude them.
- Respiratory diseases are less strongly correlated with the coal mining industry than cardiovascular diseases, for both the intercounty and intracounty analyses. This tends to work against the respiratory route as the primary source of disease.

D. CONCLUSIONS AND RECOMMENDATIONS

There is little doubt that cardiovascular and respiratory diseases in the community are somehow associated with the bituminous coal mining industry. However, the nature of that association is open to question and the results of this case study are inconclusive. The most plausible causal hypothesis is that respirable size coal dust is the etiologic agent which arrives in the community from various sources including emissions from coal preparation plants, coal transport operations, and mine ventilation systems. There is evidence that links the elevation in community disease with the proximity of the community to the thermal dryer. However, because the stack emissions alone do not appear to provide sufficient community pollution, we surmise that the source of the harmful pollution is the coal dust emissions from all the coal mining operations that center on the coal preparation plant including coal transportation and handling operations.

We have found two other possible hypotheses that link industry pollution directly with respiratory or cardiovascular diseases; however, there is no significant evidence to support these hypotheses. We conclude that these hypotheses which involve the pollutants of coal combustion products and acid mine drainage may be contributors but are not the principal cause.

In terms of study methodology, the major difficulty with the study approach was the inability to verify population exposure, either in terms of ambient pollutant concentrations or in terms of time of exposure (i.e., duration of residence). Because of the many sources of the hypothesized etiological agent and the difficulty of modeling pollutant dispersions over complex terrains, the only solution for verification of ambient concentrations is on-site measurements. In the case of retrospective studies, such as ours, the present-day measurements would have to be extrapolated to previous source conditions. Since essentially no health records include duration of residence information on them, to verify duration of exposure requires a tedious and time-consuming search of state and local records. This was too expensive an operation for the present methodological investigation; however, it is necessary for establishing the existence of a causal relationship.

A major difficulty with the investigation was the small populations found in coal mining communities. There were not enough deaths to permit the useful examination of the effects of age, year of death, specific disease, etc. Aggregation of populations over time and disease categories was necessary to be able to show statistical significance, and these aggregations obscured many of the effects that could illuminate the nature of the relationship.

With regard to further investigating the bituminous coal mining-respiratory/cardiovascular disease relationship, we recommend:

(1) *Mortality studies at several additional sites to obtain a measure of consistency.* These studies are fairly easy to perform and, in the absence of larger populations which can show the effect of significant factors, the multiple-site approach is a feasible alternative.

(2) *A concerted effort to verify coal dust exposure at promising sites for which there is a significant mortality pattern.* Extensive measurements and modeling are required to verify concentrations, and a thorough record search of decedents or phone follow-up of relatives of decedents is necessary to establish duration of exposure.

(3) *A detailed study of the differences in the smoking, diet, and heating habits between coal mining and non-coal mining communities.* This requires interviews and questionnaires.

With regard to general methodology for epidemiological investigation of environmental relationships, the following recommendations are made:

(1) Attempts should be made to standardize death certificates to include zip code, length of residence at present address, and occupation. This information should be recorded on tape to facilitate the use of the data in mortality studies.

(2) NEDS should be expanded to include the dates that processes and controls came on-line. This is important information for determining historical exposure. In addition, it would be helpful if process descriptions were provided in more detail.

(3) Centralized morbidity systems such as cancer registries and PAS should include the zip code and place of residence on patient records. Without this information, the records are not particularly useful in environmental epidemiology. Also, the occupation and duration of residence of the patient would be a great help.

Chapter VIII

VISCOSE RAYON INDUSTRY CASE STUDY

A. *INTRODUCTION*

During the selection of industry-disease associations for study, it was felt desirable to include one case study selected by a different method from that initially prescribed for the investigation. One reason was that the basic method, regression analysis by counties, might fail entirely to select useful associations. For example, the "real" associations might all be obscured because a county is too large a unit for study, or it might be that strong associations were found only in areas of intense industrialization and hence of multiple exposure, with poor prospects of disentangling the effects of specific emissions from one industry. Another reason for testing alternative methods of selection was that EPA regarded methodological development as an objective of equal stature to the search for possible specific causal relationships.

The regression analysis was a purely objective method of selection, with no preconceived associations. Accordingly, for the present study we sought an industry-disease combination where a specific association might be expected. We also sought one that offered a simple exposure to toxicants, free from confounding pollutants.

A brief review of industries and contact with public health officials soon identified the viscose rayon industry as a promising subject for study. It has an uncomplicated air pollution potential (carbon disulfide and hydrogen sulfide only); about half of the plants are in small towns or rural areas; and there is a lot of evidence about occupational health hazards, including cardiovascular disease. Community exposure was indicated by complaints about odor. Finally, a comparison of counties with viscose rayon plants and control counties without, showed elevated mortality from coronary heart disease in some of the counties with viscose rayon industry. Accordingly, we had a causal hypothesis, suitable for testing, that involved coronary heart disease and other cardiovascular effects.

Since the hypothesis was developed in this way, the viscose rayon study is different from the other three in that there was no field survey for hypothesis development. Instead, we proceeded directly to test the hypothesis at two sites.

B. DEVELOPMENT OF HYPOTHESIS

1. Selection of Viscose Rayon Industry

The first indicator that viscose rayon might be a suitable subject for study was the community complaint of odor commonly associated with the industry. It was noted that although hydrogen sulfide was undoubtedly the main cause of complaint, it was known to be accompanied by substantial amounts of carbon disulfide, possibly a greater health hazard to the community; hydrogen sulfide has a much lower odor threshold.

On closer examination of the industry, it became apparent that it offered excellent conditions for an uncluttered study of community health in the vicinity of industrial activity. About half of the U.S. viscose rayon plants are located outside of metropolitan areas, in small towns (populations 10,000 to 20,000) or in rural areas.

Selection of the industry was finally determined by the existence of considerable evidence about occupational disease and some indication of an environmental hazard; see Sections B.3 and B.4 below.

2. Description of the Viscose Process

The manufacture of viscose rayon fiber begins with sheets of bleached cellulose sulfate (from wood pulp or cotton wastes) delivered from a pulping mill. The raw cellulose is placed in a steeping press and soaked in 17% to 20% sodium hydroxide for one hour, after which excess liquor is drained off, carrying with it impurities such as cellulose degradation products.

The soft alkali cellulose remaining is shredded to flakes in a shredder, and the resulting crumbs are aged for 24 to 48 hours at 75°F in large steel vessels.

The aged crumbs are placed in large churns to begin the xanthation process in which the crumbs are churned with carbon disulfide under reduced pressure for two hours to produce small balls of cellulose xanthate:

$$R_{cell}\ ONa + CS_2 \rightleftharpoons R_{cell}\ OCSSNa \quad \text{(Xanthation Reaction)}$$

(where R_{cell} represents a cellulosic carbohydrate).

The xanthate balls are then dissolved in dilute sodium hydroxide solution to produce the viscose solution. This reaction takes two to three hours. The viscose is ripened in a series of tanks under continuous blending and deaeration during which viscose modifiers (amines and ethylene oxide polymers) may be added. Ripening is performed to adjust the concentration of sodium cellulose xanthate to proper specifications for the spinning operation:

$$R_{cell}\ OCSSNa \rightleftharpoons R_{cell}\ ONa + CS_2\uparrow \quad \text{(Ripening Reaction)}$$

The viscose solution is then injected through a spinneret die containing 750 to 2,000 minute holes into an acid spin bath containing sulfuric acid and zinc sulfate. The acid bath does the following:

- It coagulates the viscose, forming a skin around the filament which regenerates to restore cellulose. The overall reaction is as follows:

$$R_{cell}\ OCSSNa + H_2SO_4 \longrightarrow R_{cell}\ OH + Na_2SO_4 + CS_2\uparrow$$

- It yields intermediate formation of a zinc xanthate which allows more time for stretching, aligning, and compacting regenerated cellulose.
- It decomposes by-products of the xanthation process. Among the decomposition reactions are the following which give rise to significant quantities of hydrogen sulfide and carbon disulfide:

$$Na_2CS_2 + H_2SO_4 \longrightarrow Na_2SO_4 + H_2S\uparrow + CS_2\uparrow$$

$$Na_2S + H_2SO_4 \longrightarrow Na_2SO_4 + H_2S\uparrow$$

The fibers thus produced are stretched, cut, finished, wound, and shipped.

A flow diagram of the entire viscose process is presented in Figure VIII-1.

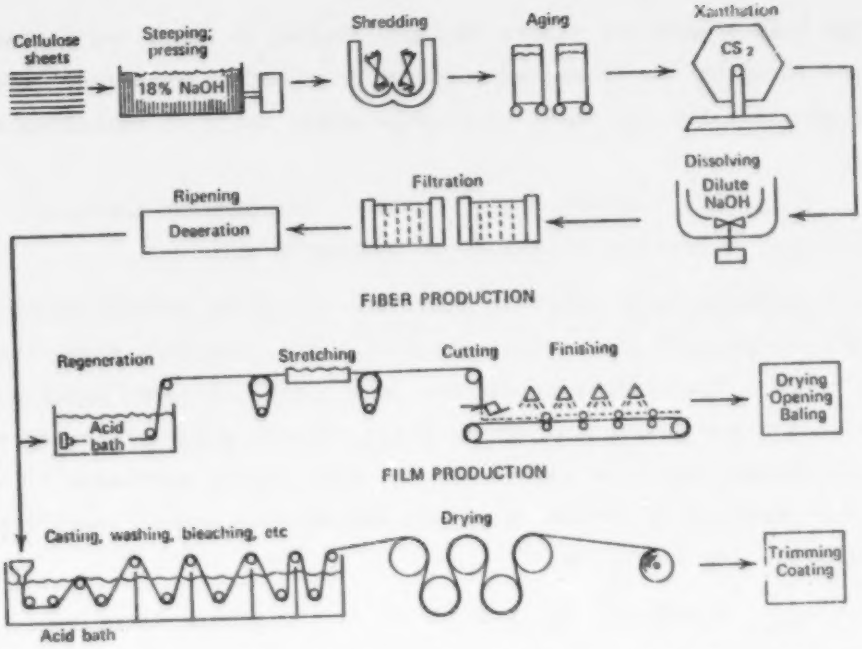

Figure VIII-1. Flow Diagram of the Viscose Process

This process description indicates process areas from which the pollutants of primary concern would emanate. Carbon disulfide is exhausted from ripening tanks and the spinning room, and hydrogen sulfide is exhausted primarily from the spinning room. Emission rates of these materials are discussed later. Although most of the gases emanating from the processes are exhausted to the atmosphere through a fume stack, some fugitive emissions would also be anticipated.

Aqueous effluents from the process result from the disposal of wastes from the recycled spin bath chemicals and from washing and other final treatment of the fiber. Untreated effluent contains zinc sulfates, sulfuric acid, sodium sulfate, carbon disulfide, hydrogen sulfide, and traces of cellulosic material lost from the process stream. Concentrations of these compounds in the effluent from the plant depend on the efficiency of the spin bath recovery cycle and the amount of treatment that wastewater receives before being discharged. Amounts of these chemicals discharged per ton of fiber produced are not available at this time; however, for every pound of regular rayon produced, 0.05 pounds of zinc sulfate and 0.84 of pure sulfuric acid are used.

The finishing of rayon fiber involves the use of large quantities of water for washing, rinsing, and bleaching. The bleach process may add low concentrations of sodium hypochlorite, hydrogen peroxide, or chlorine dioxide to the wastewater stream.

3. *Evidence of Occupational Disease*

There is substantial evidence of increased incidence of various diseases among workers in the viscose rayon industry. The occupational subgroups most affected are workers in the spinning room and in the cutting, washing, and drying areas, where exposures to carbon disulfide and hydrogen sulfide are greatest. Few studies on rayon workers have made an effort to distinguish between exposures to the two agents since the agents occur together in job areas where concentrations are greatest. A recent publication by the National Institute for Occupational Safety and Health critically evaluates a number of retrospective, prospective, and clinical epidemiologic studies done on viscose rayon workers [1]. These studies are summarized in Table M-1 in Appendix M which is adapted from that document, and reports the number of workers studied, their mean age, concentrations of toxicant, mean duration of exposure, and effects noted. Table M-2 outlines animal studies of the toxicity of carbon disulfide.

The CS_2-H_2S mixture induces cardiovascular effects, reproductive system effects, neurologic effects, effects on the eyes, and other effects. Development of atherosclerotic lesions at relatively early ages has been reported among rayon workers [2-5]; this is thought to lead to increased cardiovascular and coronary artery disease [6]. A recent review article [7] addresses toxicologic and physiologic manifestations of chronic and acute carbon disulfide exposures of animals and man. Neurologic, psychiatric, and ocular manifestations are some of the more prominent problems relating to CS_2 intoxication which are addressed. Mancuso reports on CS_2 as a cause of suicide in man [8].

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[1] National Institute for Occupational Safety and Health, *Criteria for a Recommended Standard: Occupational Exposure to Carbon Disulfide*, Washington, D.C., 1977.

[2] Tiller, J. R., R. S. F. Schilling, and J. N. Morris, Occupational toxic factor in mortality from coronary heart disease, *Br. Med. J.* *4*:407-411, 1968.

[3] Attinger, E., Chronisch Schwefelkohlen stoffvergiftung unter dem "scheinbar ungewöhnlichen" Bilde einer schweren Gefässkrankheit, *Schweiz. Med. Wochenschr.* *78*:667, 1948.

[4] Nunziante, C. A., *J. Clin. Med.* *34*:731, 1953.

[5] Browning, E., *Toxicity and Metabolism of Industrial Solvents*, Elsevier Publishing Company, Amsterdam, 1965.

Two sets of studies reported in the literature in recent years lay the foundation for the viscose rayon-cardiovascular disease association. Tiller et al. and Schilling [2,6] have reported on a retrospective mortality study in England and Wales. Hernberg and colleagues have published a series of reports [9-14] on a retrospective study and an ongoing prospective study in Finland, begun in 1967 and continuing to the present. Both sets of studies report significantly elevated mortality due to coronary heart disease in the CS_2/H_2S-exposed viscose rayon workers, compared with nonprocess workers or other sets of controls.

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[6] Schilling, R. S. F., Coronary heart disease in viscose rayon workers, *Am. Heart J. 80*:1-2, 1970.

[7] Davidson, M., and M. Feinleib, Carbon disulfide poisoning: A review, *Am. Heart J. 83*:100, 1972.

[8] Mancuso, T. F., and B. Z. Locke, Carbon disulfide as a cause of suicide—Epidemiological study of viscose rayon workers, *J. Occup. Med. 14*:595-606, 1972.

[9] Hernberg, S., T. Partanen, C. H. Nordman, and P. Sumari, Coronary heart disease among workers exposed to carbon disulphide, *Br. J. Ind. Med. 27*:313-325, 1970.

[10] Hernberg, S., C. H. Nordman, T. Partanen, V. Christiansen, and P. Virkola, Blood lipids, glucose tolerance and plasma creatinine in workers exposed to carbon disulfide, *Work, Environ. Health 8*: 11-16, 1971.

[11] Hernberg, S., M. Nurminen, and M. Tolonen, Excess mortality from coronary heart disease in viscose rayon workers exposed to carbon disulfide, *Work, Environ. Health 10*:93-99, 1973.

[12] Nurminen, M., Survival experience of a cohort of carbon disulphide exposed workers from an eight-year prospective follow-up period, *Int. J. Epidemiol. 5*:179-185, 1976.

[13] Tolonen, M., S. Hernberg, M. Nurminen, and K. Tiitola, A follow-up study of coronary heart disease in viscose rayon workers exposed to carbon disulphide, *Br. J. Ind. Med. 32*:1-10, 1975.

[14] Hernberg, S., M. Tolonen, and M. Nurminen, Eight-year follow-up of viscose rayon workers exposed to carbon disulfide, *Work, Environ. Health 2*:27-30, 1976.

In the study by Tiller et al. [2], death records were combed for factory workers who were grouped as former process workers or former nonprocess workers. A set of controls consisting of deceased nonrayon workers was selected from the same death registers. The death certificates were classified by cause, age at death, and occupation. Percentage of deaths from specific causes was the dependent variable. Results showed that percentage of deaths due to coronary heart disease was significantly higher ($p < 0.001$) in rayon process workers (42%) than in nonprocess rayon workers (24%), other local men (17%), and British men as a whole (14%). Certain defects in the study were noted, such as vagueness of the term "process" worker, lack of data on extent and length of exposure, unsuitability of control population, etc. Nevertheless, the study does show an association between rayon workers and coronary heart disease.

Hernberg and co-workers did a retrospective mortality study and a prospective morbidity and mortality study on a cohort of 343 Finnish rayon workers. For the retrospective cohort, the researchers reported that 52% of the deaths among rayon workers were attributable to coronary heart disease, while the expected percentage based on national mortality experience was 31.7%. This difference was significant at the $p < 0.002$ level for a two-tailed test [9]. Clinical parameters in the prospective study included ECG following exercise, chest radiography, blood pressure, blood lipids, glucose tolerance, and plasma creatinine and cholesterol. Of these, only blood pressure and creatinine concentration were significantly different from matched controls [10,11]. Finally, the follow-up of the cohorts of workers has led to Nurminen's life table study [12] which, based on the 8-year cumulative incidence rates of coronary heart disease for exposed and control groups, gave a relative risk of 2.2, significantly larger than 1.0.

These two sets of studies are the main basis for looking at coronary heart disease in residents near viscose rayon plants. As already noted, the tables in Appendix M summarize the range of physiologic and pathologic conditions which have been reported as possibly being related to exposure to CS_2 or CS_2/H_2S mixtures.

4. *Evidence of Environmental Exposure*

Three studies in the United States monitored hydrogen sulfide concentrations near viscose rayon plants and a Russian study monitored hydrogen sulfide and carbon disulfide. The results are summarized in Table VIII-1. The Russian study is the only one we found that measured carbon disulfide; unfortunately, sampling protocol, analytical methods, mean concentrations, and statistical analysis were not reported. For other studies, carbon disulfide concentrations were estimated on the basis of an assumed ratio to hydrogen sulfide in the emissions, as described later in the site studies (Section C).

5. *Hypothesis*

On the basis of the foregoing evidence, we decided to test the following hypothesis:

Emission of carbon disulfide and hydrogen sulfide (or one of them) from viscose rayon plants causes increased incidence of coronary heart disease and other cardiovascular disease in the exposed population.

(The site studies looked for associations also with other diseases, for two reasons: in case all diseases, or an unrelated group, were elevated by a nonspecific factor; and to look for increased incidence in other diseases known to be associated with the specific toxicants.)

Table VIII-1. *AMBIENT CS_2 AND H_2S CONCENTRATIONS IN VICINITY OF VISCOSE RAYON PLANTS*

LOCATION	SUBSTANCE	MEAN CONCENTRATION (μg/m³)	RANGE (μg/m³)	METHOD	REFERENCE
ELIZABETHTON, Tennessee					
Site 1: 610 m W of plant	H_2S	14.8	0.3-82.5	APHA bubbler method, 24-hr samples	1
Site 2: 1070 m ENE of plant	H_2S	1.2	<1-2.1		
Site 1	CS_2	~844	--	Extrapolation based on CS_2:H_2S ratio of 57:1	2
Site 2	CS_2	~68	--		
LOWLAND, Tennessee					
Site 1: 610 m SW of plant	H_2S	8.6	0.6-25.7	APHA bubbler method, 24-hr samples	1
Site 2: 1070 m NE of plant	H_2S	15.3	1.1-43.5		
Site 1	CS_2	~86	--	Extrapolation based on CS_2:H_2S ratio of 10:1	3
Site 2	CS_2	~153	--		
USSR: 910 m from factory	H_2S	--	1.4-49.5		4
	CS_2	--	5.4-222		
ENKA, North Carolina					
1.6 km W of plant	$[H_2S] > 15$ μg/m³ 234 days/365 days			Lead Acetate Tape	5
1.6 km ESE of plant	$[H_2S] > 15$ μg/m³ 46 days/365 days				
8 km W of plant	$[H_2S] > 15$ μg/m³ 151 days/365 days				

References:

1 Mincer, R. A., and K. E. Noll, University of Tennessee, Knoxville, Regulations Governing Specific Odorous Compounds, 1974.
2 Source emission data obtained through Tennessee Department of Public Health.
3 NIOSH, Occupational Exposure to Carbon Disulfide, 1977.
4 Pushkina, N. N., G. I. Korotkova, and Z. F. Chekanovskaya, Hygiene and Sanitation 35(9):476-477, 1970.
5 Resources Research, Inc., Hydrogen Sulfide Measurements Near Canton and Enka, N.C., December 1967 through November 1968.

C. TEST OF HYPOTHESIS

1. Selection of Sites

Sites were selected for hypothesis testing by surveying all towns that have had viscose rayon plants for a period of 30 or more years. Criteria for selection were:

- Elevated county mortality rates for the diseases involved in the hypothesis.
- Little or no presence of other industry which might lead to confounding emissions and/or effluents.
- Sufficient population at risk from plant emissions for an epidemiologic study.
- Existence of emission and/or ambient air data on the pollutants of interest.
- Available history of industrial activity (date of original plant construction, duration of production of viscose rayon, production capacity, etc.).

Table VIII-2 summarizes data for 15 locations.

The final selection was made by site visits to assess geographic, topographic, and demographic characteristics. Four sites were visited: Enka, North Carolina; Elizabethton, Tennessee; Lowland, Tennessee; and Lewistown, Pennsylvania. Elizabethton and Lewistown were chosen as the best sites because of the close proximity of the population to the emission source. Lewistown's plant was closed by flooding in 1972 and has not reopened. This was not a serious disadvantage because the period of operation (1921-1972) was appropriate to our study. It also offered the possibility of detecting a downtrend in mortality or morbidity; however, this could not be done with the available data.

Table VIII-2. *VISCOSE RAYON-PRODUCING TOWNS AND THEIR APPROPRIATENESS FOR EPIDEMIOLOGIC STUDY*

TOWN, STATE	COUNTY	WMMR > NMMR + 2σ †	WFMR > NMMR + 2σ †	INDUSTRIAL CLUTTER	POPULATION POTENTIALLY AT RISK	EMISSION INFORMATION	PLANT LOCATION	DATE OF ORIGINAL PLANT CONSTRUCTION
Enka, North Carolina	Buncombe	No	No	Medium	60,000	Yes	Suburban	1929
Elizabethton, Tennessee	Carter	Yes	No	Low	12,000	Yes	Urban	1925
Lowland, Tennessee	Hamblen/Green	Yes	Yes	Medium	>1,000	Yes	Rural	1948
Front Royal, Virginia	Warren	Yes	No	Low	8,000	Yes	Urban	1941
Lewistown, Pennsylvania	Mifflin	Yes	Yes	Low	11,000	No	Urban	1921
Childersberg, Alabama	Talladega	No	Yes	Medium	5,000			
	Shelby	No	No	Medium				1949
Fairhaven, Vermont, New York	Rutland (Vt.)	No	No	Low	3,000			
	Washington, (N.Y.)	Yes	Yes	Low				
Painesville, Ohio	Lake	Yes	Yes	Medium	22,000			
Utica, New York	Oneida/Herkimer	Yes	Yes	High	90,000			1969
Fredericksburg, Virginia	Stafford/ Spotsylvania			High	15,000			1936
Nitro, West Virginia	Putnam/Kanawha			High	8,000			1927
Parkersburg, West Virginia	Kanawha			High	44,000			1952
LeMoyne, Alabama	Mobile			High	190,000			

† WMMR = White male mortality rate for myocardial infarction
NMMR = National mean mortality rate for myocardial infarction
WFMR = White female mortality rate for myocardial infarction

2. *Elizabethton Study*

a. *Site Description*

The Beaunit Fibers plant in Elizabethton, Tennessee, commenced operation in the mid-1920's. It has produced rayon by the viscose process from that time up to the present. A neighboring yarn plant (Bemberg) emitted detectable quantities of ammonia into the atmosphere from the cuprammonium process. This plant was shut down in 1975.

Elizabethton is located in Carter County, Tennessee, which is in the northeast corner of the state. It is a town of about 12,000 population located at the confluence of the Doe and Watauga Rivers. The Beaunit and Bemberg plants are west of the center of town. Two to three hundred homes in Elizabethton are 600 to 2000 meters south of the plant. The remainder of the population lives 0.8 to 4 kilometers east of the plant.

The Beaunit and Bemberg plants are located in the Watauga River Valley about 1500 feet above sea level (see Figure VIII-2). Hills surrounding the plants range from 300 to 600 feet above the plant. Data in Section b below indicate that annual average concentrations of carbon disulfide would be on the order of 130 to 450 $\mu g/m^3$ in Elizabethton. The entire population of Elizabethton is postulated to be at risk as evidenced by the transport analysis in the following section.

b. *Population Exposure*

Exposure of the Elizabethton population to plant emissions can be estimated in two ways:

- From field measurements of hydrogen sulfide (Mincer and Noll, 1974; see Table VIII-1 above). These showed means of 14.8 and 1.2 $\mu g/m^3$ in the vicinity of the plant. Applying a ratio of $CS_2:H_2S$ = 57:1 (see below), this implies mean carbon disulfide concentrations of 68 to 844 $\mu g/m^3$ (in the conditions of the field tests).
- From estimated emission rates, used as source strengths in a diffusion model. Estimated rates are shown in Table VIII-3. The ratio $CS_2:H_2S$ ranges from 2:1 to 57:1, and we chose to use the figure from the Elizabethton plant, i.e., 57:1.

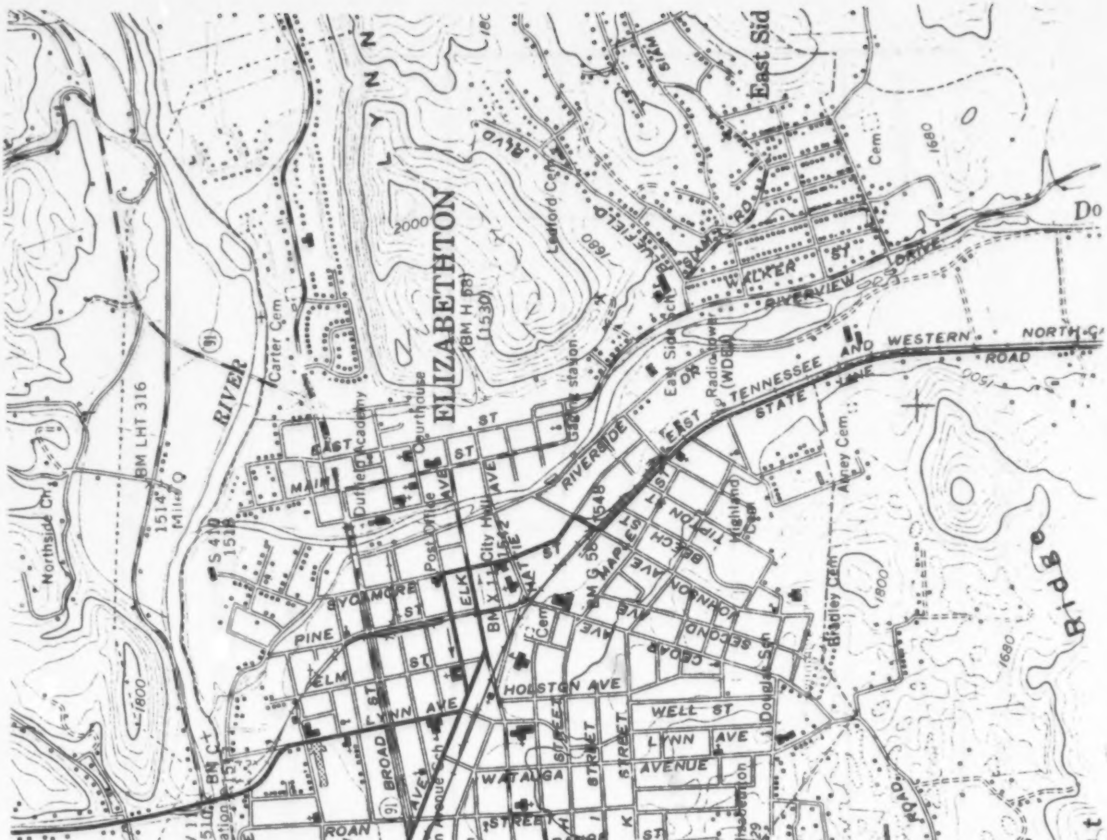

Figure VIII-2. Map of Elizabethton, Tennessee

Table VIII-3. CS_2 AND H_2S EMISSION INFORMATION FROM VISCOSE RAYON PLANTS

SITE/SOURCE	EMISSIONS (lb/ton rayon)		$CS_2:H_2S$ RATIO	METHOD	REFERENCE
	CS_2	H_2S			
Elizabethton, Tennessee (Beaunit Fibers)	960	16.75	57:1	Engineering Calculation	1
Enka, North Carolina (American Enka)	551	120	5:1	Engineering Calculation	2
	300	54	6:1	Analysis of Emission Points	
EPA	55	6	9:1	Engineering Calculation (reliability rating: E)	3
NIOSH			2:1 to 10:1		4

References:

1 Source emission data obtained through Tennessee Department of Public Health; also contained in EPA's National Emissions Data System.

2 Personal correspondence, C. T. Mitchell, American Enka Company.

3 EPA, Air Pollutant Emission Factors, AP-42

4 NIOSH, Criteria for a Recommended Standard: Occupational Exposure to Carbon Disulfide, p. 23, 1977.

The diffusion calculations were developed as described in Appendix A, which presents a method of modeling long-term concentrations of pollutants based on emission characteristics of the plant and local meteorology. Turner's model [15], equation (4) in the appendix, was calculated using the input data presented in Table VIII-4. The plume rise equation according to Holland was used to calculate *H* (see Appendix A, p. A-3). The relative frequency function was obtained from a STAR program (National Climatic Center,Asheville, North Carolina) run for Tri-City Airport, 13.5 miles from Elizabethton, and based on observations from January of 1966 to December of 1970, 8 observations per day. This particular STAR was run for only five Pasquill stability classes: three unstable conditions, the neutral condition, and an aggregate of all stable conditions.

Long-term concentrations of CS_2 were calculated for hypothetical receptors at 0.25, 0.5, 0.75, 1, 1.5, 2, 2.5, 3, 4, 5, and 10 kilometers from the plant in 16 directions (points of the compass)(see Table VIII-5).

Radial grid lines were superimposed on a topographic map of Elizabethton and the calculated concentrations placed on the grid at appropriate distances and directions. Analysis centered on concentrations in the eight primary directions (N, NE, E, SE, S, SW, W, NW) since there is slight bias in reporting of wind direction that is partial to these directions. Topographic considerations were next addressed. The pollutant concentration was assumed to be greater due to wind flow patterns in valleys and less on mountains and ridges. The resulting concentration isopleths can be seen in Figure VIII-3. It must be emphasized that the intention is only to show that significant exposure is not inconsistent with available data. An extensive field study, with a grid of sampling stations and on-site meteorological observations over a year or more, would be needed for reliable quantitative estimates of population exposure. However, it appears that the calculated estimates may be of the right order of magnitude. Table VIII-1 shows a mean H_2S

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[15] Turner, D. B., *Workbook of Atmospheric Dispersion Estimates*, PHS Publication 999-AP-26, 1969.

Table VIII-4

INPUT PARAMETERS FOR HOLLAND PLUME RISE EQUATION AND TURNER MODEL

Parameter	Value	Reference
Physical stack height	12.2 m	1
Stack gas exit velocity	15.24 m/sec	1
Inside stack diameter	0.66 m	1
Stack gas temperature	302°K	1
Ambient temperature	287.1°K	2
Average wind speed	1.5 m/sec	-
Atmospheric pressure	1.013×10^3 millibars	-
Effective stack height	22.85 m	3
Relative frequency function	STAR routine (Tri-City Airport, TN)	4
CS_2 emission rate	112 g/sec	1
Concentrations determined at distances of 0.25, 0.5, 0.75, 1, 1.5, 2, 2.5, 3, 4, 5, and 10 kilometers		-
Standard deviation of vertical component of plume determined for appropriate distance for appropriate stability class		5

References:

1 Tennessee Department of Public Health, Source Emission Data for Beaunit Plant, Elizabethton, Tenn., 1975

2 U.S. Department of Commerce, Environmental Science Services Administration, Climatic Atlas of the United States, 1978.

3 Calculated from above data after the method of Holland (see Appendix A).

4 STAR Tabulation, Bristol, Tenn., National Climatic Center, Asheville, N.C., 1971.

5 Turner, D. B., Workbook of Atmospheric Dispersion Estimates, PHS Publication 999-AP-26, 1969.

Table VIII-5

CALCULATED LONG-TERM CONCENTRATIONS OF CARBON DISULFIDE EMISSIONS AT VARIOUS DISTANCES FROM BEAUNIT FIBERS PLANT, ELIZABETHTON, TENNESSEE

Wind Direction	Estimated Long-Term Concentrations of CS_2 ($\mu g/m^3$)										
	Distance (km)										
	0.25	0.5	0.75	1	1.5	2	2.5	3	4	5	10
N	199	223	170	127	76	51	37	28	19	13	5
NNE	215	275	221	169	103	70	51	39	26	19	7
NE	346	578	491	383	238	162	118	91	60	44	16
ENE	404	596	489	375	230	156	114	88	58	42	15
E	578	649	503	378	227	153	110	85	56	40	14
ESE	276	274	207	154	92	62	44	34	22	16	6
SE	152	138	101	74	44	29	21	16	11	8	3
SSE	99	106	82	61	37	25	18	14	9	6	2
S	237	234	172	126	75	50	36	28	18	13	5
SSW	158	169	130	97	59	39	28	22	14	10	4
SW	322	390	299	223	134	90	65	50	33	23	8
WSW	330	374	275	201	119	79	57	43	28	20	7
W	423	460	335	244	143	95	68	52	34	24	8
WNW	277	307	226	165	97	65	47	36	23	17	6
NW	305	244	166	117	66	43	31	23	15	11	4
NNW	110	102	75	55	32	22	16	12	8	6	2

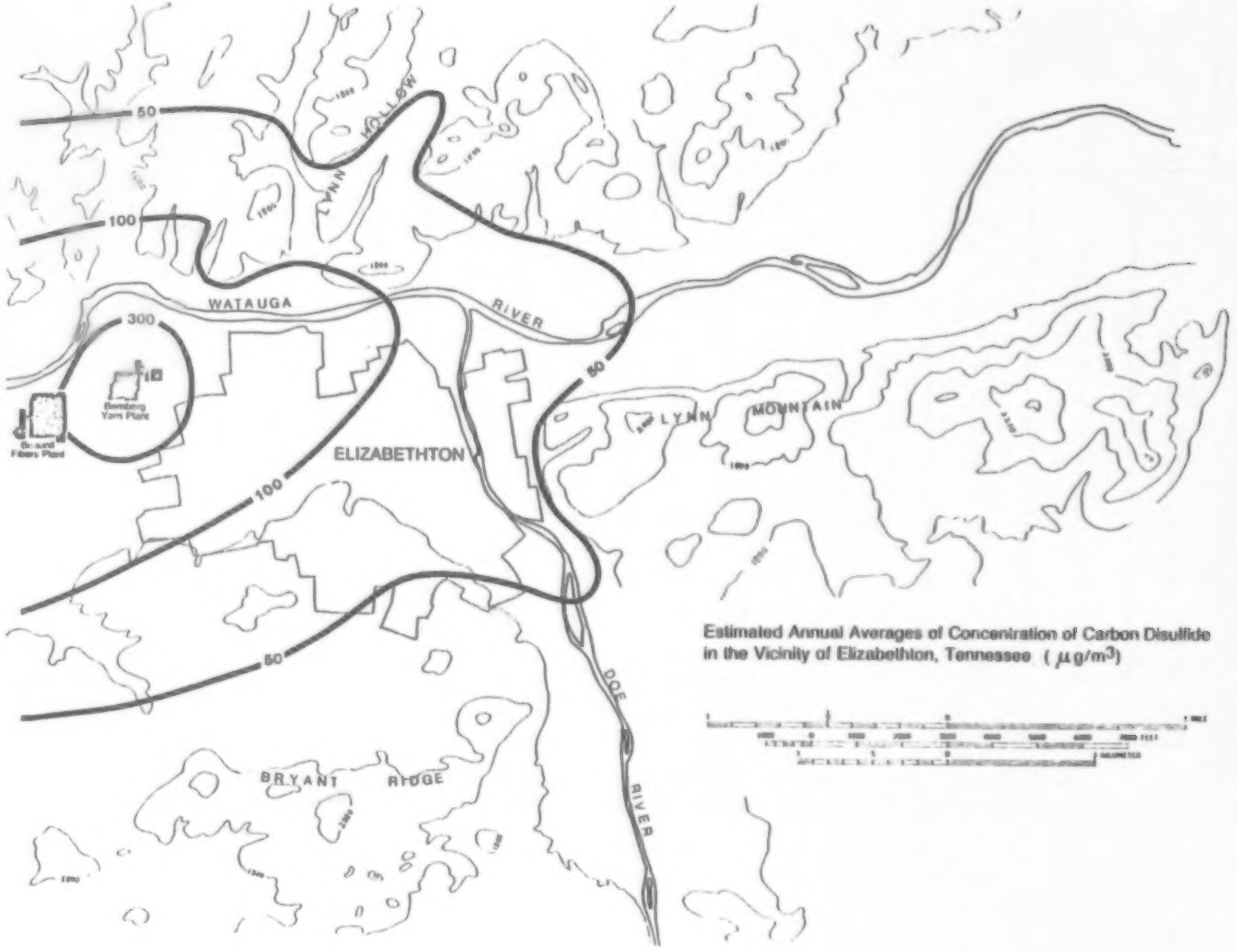
Estimated Annual Averages of Concentration of Carbon Disulfide in the Vicinity of Elizabethton, Tennessee ($\mu g/m^3$)

Figure VIII-3. Concentration Isopleths: Elizabethton, Tennessee

concentration of 14.8 $\mu g/m^3$ at 610 m W of the plant, and 1.2 $\mu g/m^3$ at 1070 m ENE. Applying the CS_2:H_2S ratio of 57:1 (see Table VIII-3), the estimated CS_2 concentrations are 844 $\mu g/m^3$ and 68 $\mu g/m^3$. Interpolation in Table VIII-5 gives concentrations of 600 $\mu g/m^3$ and 200 $\mu g/m^3$ at these points.

c. *Mortality*

Age-adjusted mortality rates (standardized to the 1970 U.S. population distribution) were computed for deaths occurring between 1965 and 1975. Table VIII-6 presents these data and rate ratios for Elizabethton white males and two sets of controls (i.e., (1) Carter County excluding Elizabethton and (2) Athens, Newport, and Greenville). Table VIII-6 presents the same data for white females.

It is apparent from Table VIII-6 that cardiovascular disease mortality is not elevated in Elizabethton as compared with its controls. In fact, among males, only cancer of the urinary bladder appears elevated in Elizabethton when compared to both sets of controls. The only other causes of death which show appreciable elevation compared to either control are cancers of the oral cavity, pharynx, esophagus, stomach, and pancreas, and other diseases of the urinary system. None of these rate ratios exceeds 2.4, and their significance, both statistically and practically, is dubious.

Among white females, diseases of the liver, gallbladder and pancreas; bronchitis, emphysema and asthma; and cancer of the colon or rectum are elevated in Elizabethton compared to both controls. Rate ratios for these diseases range from 2.0 to 4.0. Cardiovascular mortality, with which we are particularly interested, shows no elevation in Elizabethton and, if anything, appears in excess in the controls.

Table VIII-6. AGE-ADJUSTED MORTALITY RATES (PER 100,000 POPULATION), 1965-1975, ELIZABETHTON AND CONTROLS: WHITE MALES, WHITE FEMALES

CAUSE OF DEATH	WHITE MALES MORTALITY RATES					WHITE FEMALES MORTALITY RATES				
	Elizabethton	Control #1	Rate Ratio	Control #2	Rate Ratio	Elizabethton	Control #1	Rate Ratio	Control #2	Rate Ratio
Infectious & parasitic diseases	12	7	1.7	16	0.8	3	3	1.0	5	0.6
Cancer, oral, pharynx & esophagus	28	6	4.7	36	0.8	3	9	0.3	9	0.3
Cancer, stomach	22	9	2.4	25	0.9	6	14	0.4	8	0.8
Cancer, duodenum & small intestine	0	0	-	2	0	0	1	0	1	0
Cancer, colon & rectum	37	30	1.2	21	1.8	54	18	3.0	23	2.3
Cancer, liver & bile ducts	3	5	0.6	6	0.5	9	6	1.5	5	1.8
Cancer, pancreas	15	7	2.1	26	0.6	0	4	0	14	0
Other digestive cancers	0	0	-	2	0	0	2	0	0	-
Cancer, respiratory organs	125	113	1.1	140	0.9	20	18	1.1	21	1.0
Cancer, genitourinary organs except bladder	44	26	1.7	26	1.7	26	63	0.4	51	0.5
Cancer of the bladder	18	1	18.0	3	6.0	3	6	0.5	5	0.6
Other cancers except leukemia	42	53	0.8	47	0.9	73	60	1.2	73	1.0
Neoplasms of lymphatics	33	39	0.8	35	0.9	18	15	1.2	14	1.3
Endocrine, nutritional, metabolic & blood diseases	38	35	1.1	52	0.7	27	53	0.5	48	0.6
Mental & nervous system diseases	34	22	1.5	23	1.5	3	9	0.3	22	0.1
Rheumatic heart disease	4	15	0.3	5	0.8	13	15	0.9	14	0.9
Hypertensive disease	8	19	0.4	56	0.1	3	11	0.3	32	0.1
Cardiovascular diseases	881	895	1.0	1263	0.7	349	401	0.9	450	0.8
Cerebrovascular diseases	148	134	1.1	174	0.9	107	143	0.7	108	1.0
Respiratory diseases	87	87	1.0	87	1.0	6	38	0.7	28	0.2
Bronchitis, emphysema & asthma	45	68	0.7	42	1.1	24	12	2.0	6	4.0
Pneumoconiosis	7	5	1.4	0	0	0	0	-	0	-
Other digestive diseases	0	0	-	2	0	0	0	-	1	0
Diseases of stomach & duodenum	16	20	0.8	21	0.8	6	6	1.0	3	2.0
Appendicitis, hernia & others	7	9	0.4	16	0.4	3	6	0.5	10	0.3
Gastroenteritis, noninfectious	0	0	-	2	0	0	0	-	1	0
Diseases of liver, gallbladder & pancreas	28	34	0.8	40	0.7	34	9	3.8	15	2.3
Diseases of urinary system	42	20	2.1	28	1.5	24	13	1.8	16	1.5
Diseases of genital organs	0	8	0	6	0	0	0	-	0	-
Diseases of pregnancy & childbirth	0	0	-	0	-	0	0	-	0	-
Diseases of skin & musculoskeleton & ill-defined diseases	15	15	1.0	39	0.4	25	18	1.4	15	1.6
Congenital anomalies	0	2	0	3	0	7	7	1.0	3	2.3
Perinatal diseases	0	0	-	0	-	0	0	-	0	-
Accidents, poisons & violence	136	139	1.0	142	1.0	28	54	0.5	34	0.8

Control #1: Carter County Control #2: Athens, Newport, Greenville

3. *Lewistown Study*

a. *Site Description*

Viscose rayon production was brought to the United States by the British textile firm of Courtaulds & Co., Ltd., in the 1910's. The American company, called the American Viscose Corporation, built its third plant in Lewistown, Pennsylvania. This plant began producing rayon fiber in August of 1921. During the Second World War, the American Viscose Corporation was nationalized and sold to American investors by the British Government. FMC Corporation obtained control of the five or so plants formerly owned by American Viscose in the late 1940's. In 1975, FMC sold its plants to Avtex Fibers, Inc.

The production of viscose rayon at the Lewistown plant was abruptly ended in the summer of 1972 when Hurricane Agnes caused the Juniata River to crest above the plant grounds, ruining machinery and stopping production. Since the demand for rayon was not great at that time, FMC decided not to reopen the plant.

Lewistown is located at a bend in the Juniata River. The dormant American Viscose plant is directly across the river from the center of town. The plant is less than a mile from the majority of the 11,000 residents of Lewistown who live along the bend in the river (see map, Figure VIII-4). Major topographic features influencing the air movement in the Juniata Valley include Shade Mountain to the east, Jack's Mountain to the north, and a small peak directly behind the plant.

EPA's Environmental Photographic Interpretation Complex (EPIC) provided some input into the Lewistown study. EPIC personnel surveyed aerial photographs of Lewistown taken in 1938 and 1964. Overlays were constructed, outlining residential and commercial centers in both of those years. Factories, stacks, and sewage treatement facilities were also delineated and noted on maps at the appropriate time intervals. There was no indication of substantial changes in the plant or in population distribution, suggesting that the Lewistown situation has been fairly stable over a long period.

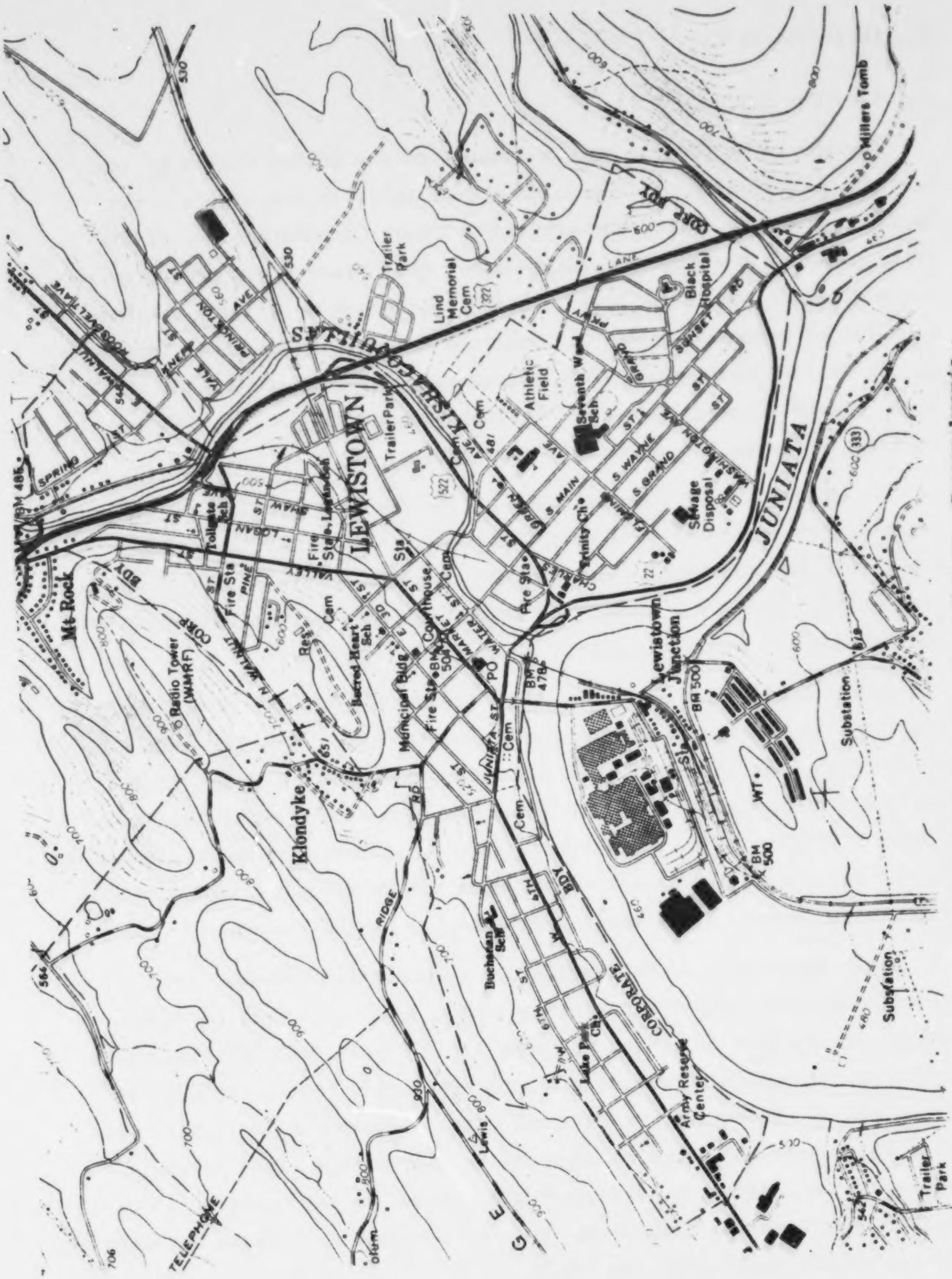

Figure VIII-4. Map of Lewistown, Pennsylvania

b. Population Exposure

It was not possible at Lewistown to establish a plausible estimate of air pollutant transport and population exposure as for Elizabethton. No ambient air monitoring had been done, and the meteorological data were judged to be quite inadequate to support theoretical estimates. Since it is now impossible to measure local concentrations of CS_2 and H_2S (unless the plant is reactivated), we are dependent on general appraisal of the likelihood of significant population exposure. The higher exhausting of fumes at Lewistown (350-foot stacks) would considerably reduce exposure close in to the plant, but would not be expected to protect more distant residents from some exposure. In fact, a 1966 report [16] on a study of pollution from the American Viscose factory states that, "You can smell them [the plant] every once in a while." Since the odor threshold for H_2S, the more odorous of the two compounds, is approximately 30 $\mu g/m^3$ [17], the implication is that during these episodes the CS_2 concentration was on the order of a few hundred $\mu g/m^3$.

c. Mortality

As was done for Elizabethton, age-adjusted mortality rates for 34 disease categories for Lewistown were compared with two sets of controls. In one analysis, rates from1965-1972 were compared to those for Mifflin County excluding Lewistown (Control #1). In the second analysis, rates for 1965-1975 were compared with those for the aggregate population of Berwick, Shamokin, Mt. Carmel, Chambersberg, and Sunbury. Age-adjusted mortality rates and rate ratios are presented in Table VIII-7 for white males and in Table VIII-8 for white females.

It can be readily seen from Table VIII-7 that mortality from cardiovascular disease is not elevated in Lewistown white males as compared to its controls. In fact, for congenital anomalies only do Lewistown males

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[16] Premerlani, R., *A Case Study of American Viscose Division of FMC Corporation of Lewistown, Pa.*, December 1966.

[17] National Institute for Occupational Safety and Health, *Criteria for a Recommended Standard: Occupational Exposure to Hydrogen Sulfide*, Washington, D.C., May 1977.

Table VIII-7

AGE-ADJUSTED MORTALITY RATES (PER 100,000 POPULATION), LEWISTOWN AND CONTROLS: WHITE MALES

CAUSE OF DEATH	MORTALITY RATES		Rate Ratio	MORTALITY RATES		Rate Ratio
	Lewistown 1965-1972	Control #1		Lewistown 1965-1975	Control #2	
Infectious & parasitic diseases	11	2	5.5	11	23	0.5
Cancer, oral, pharynx & esophagus	11	10	1.1	11	27	0.4
Cancer, stomach	11	7	1.6	11	16	0.7
Cancer, duodenum & small intestine	0	2	0	0	1	0
Cancer, colon & rectum	52	39	1.3	63	65	1.0
Cancer, liver & bile ducts	4	5	0.8	4	9	0.4
Cancer, pancreas	27	12	2.3	31	18	1.7
Other digestive cancers	0	5	0	0	1	0
Cancer, respiratory organs	111	62	1.8	157	137	1.1
Cancer, genitourinary organs except bladder	30	14	2.1	41	32	1.3
Cancer of the bladder	0	3	0	0	8	0
Other cancers except leukemia	45	28	1.6	64	64	1.0
Neoplasms of lymphatics	33	29	1.1	40	29	1.4
Endocrine, nutritional, metabolic & blood diseases	53	25	2.1	60	42	1.4
Mental & nervous system diseases	26	7	3.7	26	27	1.0
Rheumatic heart disease	8	16	0.5	15	29	0.5
Hypertensive disease	23	12	1.9	23	30	0.8
Cardiovascular diseases	974	702	1.4	1266	1197	1.1
Cerebrovascular diseases	136	84	1.6	173	167	1.0
Respiratory diseases	67	74	0.9	100	117	0.9
Bronchitis, emphysema & asthma	22	20	1.1	47	49	1.0
Pneumoconiosis	8	7	1.1	8	121	0.1
Other digestive diseases	0	0	-	0	0	-
Diseases of stomach & duodenum	12	4	3.0	12	14	0.9
Appendicitis, hernia & others	15	9	1.7	23	28	0.8
Gastroenteritis, noninfectious	0	0	-	0	0	-
Diseases of liver, gallbladder & pancreas	24	6	4.0	31	67	0.5
Diseases of urinary system	19	18	1.1	19	20	1.0
Diseases of genital organs	0	2	0	0	7	0
Diseases of pregnancy & childbirth	0	0	-	0	0	-
Diseases of skin & musculoskeleton & ill-defined diseases	4	7	0.6	7	12	0.6
Congenital anomalies	4	0	-	4	1	4.0
Perinatal diseases	0	0	-	0	0	-
Accidents, poisons & violence	106	81	1.3	120	92	1.3

Control #1: Mifflin County (excluding Boroughs of Lewistown and Juniata Terrace and Derry Township); 1965-1972.

Control #2: Berwick, Shamokin, Mt. Carmel, Chambersberg, Sunbury; 1965-1975.

Table VIII-8

AGE-ADJUSTED MORTALITY RATES (PER 100,000 POPULATION), LEWISTOWN AND CONTROLS: WHITE FEMALES

CAUSE OF DEATH	MORTALITY RATES		Rate Ratio	MORTALITY RATES		Rate Ratio
	Lewistown 1965-1972	Control #1		Lewistown 1965-1975	Control #2	
Infectious & parasitic diseases	3	13	0.2	3	5	0.6
Cancer, oral, pharynx & esophagus	9	2	4.5	9	2	4.5
Cancer, stomach	0	0	-	0	8	0
Cancer, duodenum & small intestine	0	0	-	0	1	0
Cancer, colon & rectum	28	24	1.2	33	50	0.7
Cancer, liver & bile ducts	0	9	0	3	9	0.3
Cancer, pancreas	8	6	1.3	10	10	1.0
Other digestive cancers	0	0	-	0	2	0
Cancer, respiratory organs	14	7	2.0	19	19	1.0
Cancer, genitourinary organs except bladder	45	25	1.8	53	62	0.9
Cancer of the bladder	0	2	0	2	2	1.0
Other cancers except leukemia	93	63	1.5	120	115	1.0
Neoplasms of lymphatics	17	17	1.0	24	24	1.0
Endocrine, nutritional, metabolic & blood diseases	25	19	1.3	33	58	0.6
Mental & nervous system diseases	3	8	0.4	8	7	1.1
Rheumatic heart disease	12	13	0.9	12	27	0.4
Hypertensive disease	11	22	0.5	20	17	1.2
Cardiovascular diseases	302	452	0.7	404	518	0.8
Cerebrovascular diseases	49	91	0.5	87	115	0.8
Respiratory diseases	15	28	0.5	17	21	0.8
Bronchitis, emphysema & asthma	0	5	0	9	9	1.0
Pneumoconiosis	0	0	-	0	1	0
Other digestive diseases	0	0	-	0	0	-
Diseases of stomach & duodenum	5	2	2.5	9	7	1.3
Appendicitis, hernia & others	2	16	0.1	5	16	0.3
Gastroenteritis, noninfectious	0	0	-	0	0	-
Diseases of liver, gallbladder & pancreas	17	6	2.8	29	28	1.0
Diseases of urinary system	18	8	2.3	18	8	2.3
Diseases of genital organs	2	0	0	2	2	1.0
Diseases of pregnancy & childbirth	0	0	-	0	0	-
Diseases of skin & musculoskeleton & ill-defined diseases	6	4	1.5	9	9	1.0
Congenital anomalies	2	2	1.0	2	3	0.7
Perinatal diseases	0	0	-	0	0	-
Accidents, poisons & violence	35	21	1.7	51	36	1.4

Control #1: Mifflin County (excluding Boroughs of Lewistown and Juniata Terrace and Derry Township); 1965-1972.

Control #2: Berwick, Shamokin, Mt. Carmel, Chambersberg, Sunbury; 1965-1975.

exceed *both* sets of controls. Among the causes for which Lewistown white males appreciably exceed either control are infectious and parasitic diseases, cancers of the pancreas and genitourinary system, mental and nervous system diseases, and diseases of the liver, gallbladder, and pancreas.

Deaths from cancers of the oral cavity, pharynx, or esophagus among white females are more frequent in Lewistown than in either control area. In both analyses, the rate ratio was 4.5. Also elevated when compared to both controls was the category "diseases of the urinary system," with rate ratios of 2.3. Mortality from diseases of the stomach, duodenum, liver, gallbladder, and pancreas was elevated in Lewistown as compared to the rest of Mifflin County, although the rate ratio did not exceed 2.8.

4. Discussion of Results

Ultimately, the plausibility of an industry-community disease hypothesis is dependent on the "exposed" community exhibiting higher-than-expected mortality from diseases which have been etiologically and meteorologically associated with the industry.

One can immediately see that mortality from cardiovascular disease, which has been linked etiologically to the viscose rayon industry, is not elevated in either study site for either sex. This result in males is somewhat surprising, although the occupational mortality (probably dependent on heavy exposure) would not be expected to contribute much to the mortality in Elizabethton or Lewistown.

Turning to other diseases, it appears that there is an excess of diseases of the liver, gallbladder, and pancreas among females in both study sites. Cirrhosis would seem to predominate in this disease group, and it is unlikely that the excess is environmental in nature. It may be reasonable to investigate further the apparent bladder cancer excess found in Elizabethton, as the disease has a strong occupational and environmental component.

On the basis of the industrial, demographic, and environmental characteristics of Elizabeth and Lewistown, we would expect both to show similar excesses of industry-related mortality if any did, in fact, exist. These patterns are not evident, however, particularly with respect to cardiovascular disease. It is, therefore, unlikely that the viscose rayon industry can be hypothesized as contributing to cardiovascular disease mortality in communities containing the industry.

D. CONCLUSIONS

The following conclusions have been derived from the two field investigations of the viscose rayon industry.

(1) Carbon disulfide and hydrogen sulfide are emitted from viscose rayon plants and are present in the ambient air of nearby populated areas.

(2) A hypothesis linking emissions (CS_2, H_2S) from viscose rayon plants to cardiovascular disease mortality in nearby populations is not supported by the mortality data available for 1965-1975.

(3) Further study should be made of bladder cancer in males working in the Elizabethton plant and of diseases of the liver, gallbladder, and pancreas in females in both Lewistown and Elizabethton.

Appendix A

ATMOSPHERIC TRANSPORT

Appendix A

ATMOSPHERIC TRANSPORT

INTRODUCTION

By far the most satisfactory way of estimating population exposure to air pollutants from industrial activity is from actual long-term sampling at stations throughout the area. These measurements are rarely available, and there are few to support our case studies. It is still possible to make order of magnitude estimates to test the plausibility of a hypothesized pathway from source to population at risk, by using one of the established dispersion models. This is what we have done, and this Appendix discusses the procedures.

We wish to emphasize here the limitations that we discuss at the end of the Appendix. Accurate estimates of ground level concentration from source strength and meteorological data are not easy to make, even in uncomplicated terrain. They require frequent source sampling and long-term micrometeorological observations at several stations. We have had to rely in general on guesses for source strength and on meteorological data from one or two stations 10 to 200 miles away. The best that can be expected from such calculations is an indication that one's hypothesis is not inconsistent with climatological data.

Equations used to estimate long-term average concentrations from point sources are generally similar in form. Several of the models which are used in the case studies use the long-term concentration estimates from equations similar to those developed in this Appendix. The models then go on to perform other manipulations on the data. Input data were acquired and equations were solved to give estimates of exposure. Input parameters for the long-term concentration equation are source-specific stack and emission data and meteorological data. The meteorological data have been manipulated in a STability ARray (STAR) program as discussed by Doty and Wallace [1]. The output of the STAR program is a frequency of occurrence and relative frequency distribution of various winds by wind direction, wind speed, and stability class. The relative frequency is inputted into each of the various dispersion models. Models which are used in various chapters of the report include the following:

- Air Quality Display Model (AQDM) [2]

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[1] Doty, S. R., and B. L. Wallace, *A Climatological Analysis of Pasquill Stability Categories based on 'STAR' Summaries*, NOAA, National Climatic Center, April 1976.

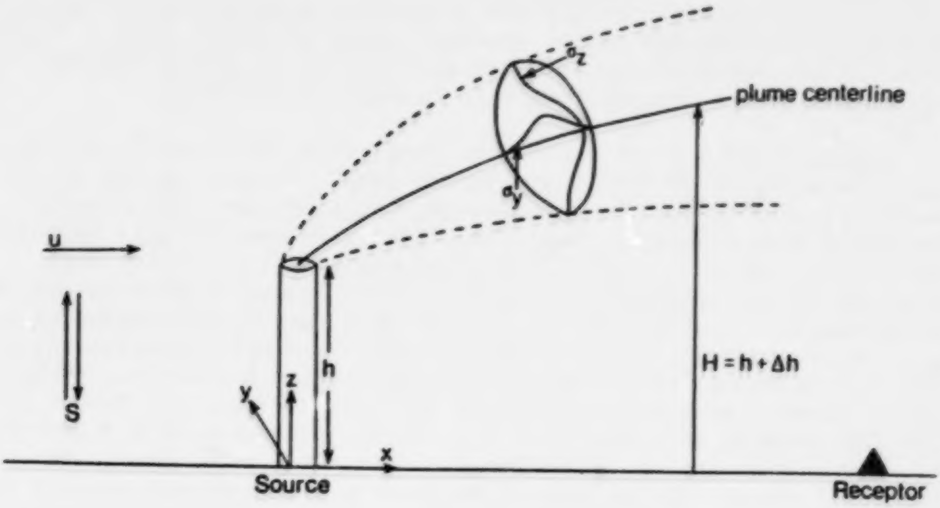
[2] TRW Systems Group, Air Quality Display Model, PB 189 194, November 1969.

- Climatological Dispersion Model (CDM) [3]
- Long-term average concentrations calculated after the work of Turner [4] and Gifford [5]

The AQDM and CDM are similar models which are used by EPA for long-term pollution potential; the CDM is an updated version of the AQDM. All of the dispersion models above contain common features which are discussed in this appendix.

DEVELOPMENT OF EQUATIONS USED IN REPORT

The Pasquill-Gifford plume formulation is the basis for the aforementioned models. This formulation is based on the dispersion of a plume as it spreads out from a stack. The diagram below illustrates the principles used in this type of dispersion modeling.



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[3] Busse, A. D., and J. R. Zimmerman, *User's Guide for the Climatological Dispersion Model*, National Environmental Research Center, EPA-R4-73-024, December 1973.

[4] Turner, D. B., *Workbook of Atmospheric Dispersion Estimates*, PHS Publication 999-AP-26, 1969.

[5] Gifford, F. A., Ch. 3, pp. 65-116, in U. S. Atomic Energy Commission, *Meteorology and Atomic Energy*, 1968.

Several features should be noted about the source-receptor relationship indicated in the above diagram:

- The source and any point in the plume may be identified by a set of x,y,z Cartesian coordinates with their origin at the base of the stack.
- The effective height of emission (H) is the height at which the plume centerline becomes horizontal. It is the stack height (h) plus an incremental factor (Δh) related to the stack parameters, buoyancy and vertical momentum of the stack effluent. The incremental factor used to determine effective height of emission in most of the case studies in this report was determined according to the method of Holland [6]:

$$\Delta h = \frac{v_s d}{u}\left(1.5 + 2.68 \times 10^{-3}\ p\ \frac{T_s - T_a}{T_s}\ d\right)$$

where:

Δh = the rise of the plume above the stack, m

d = the inside stack diameter, m

v_s = stack velocity, m sec^{-1}

u = wind speed, m sec

p = atmospheric pressure, mb

T_s = stack gas temperature, °K

T_a = air temperature, °K

and 2.68×10^{-3} is a constant having units of $\text{mb}^{-1}\text{m}^{-1}$.

The study on copper smelters used the plume rising according to Briggs [7].

- Within the plume, a Gaussian distribution of stack effluent is assumed. The limits of the plume dispersion at any distance, x, are represented by one standard deviation in the y- and z-directions (σ_y,σ_z) at that distance. At some point, L, a limit of dispersion in the z-direction is reached; this is called the depth of the mixing layer and represents the limiting value of σ_z.

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[6] Holland, J. Z., A meteorological survey of the Oak Ridge Area, pp. 554-559, in *Atomic Energy Commission Report ORO-99*, Washington, D.C., 1953.

[7] Briggs, G. A., *Plume Rise*, USAEC Critical Review Series, TID-25075, National Technical Information Service, Springfield, Virginia, 1969.

- Wind speed (*u*) and atmospheric turbulence (*S*) are important parameters affecting the plume.

- The concentration of material from the plume (χ) affecting a receptor on the ground at distance *x* and on the plume axis may be determined by solving the equation

$$\chi(x,y,z;H) = \frac{Q}{\sigma_y \sigma_z \pi u}\, exp\left[-\frac{1}{2}\,\frac{y}{\sigma_y}\right] exp\left[-\frac{1}{2}\left(\frac{H}{\sigma_z}\right)^2\right] \qquad (1)$$

for $y,z = 0$,

$$\chi(x,0,0;H) = \frac{Q}{\sigma_y \sigma_z \pi u}\, exp\left[-\frac{1}{2}\left(\frac{H}{\sigma_z}\right)^2\right] \qquad (2)$$

The long-term effect of wind direction frequency distribution is dealt with as follows: The circumference at distance χ is divided into 16 sectors, each corresponding to a wind vector which blows toward it. The sector width is then $2\pi x/16$. If it is assumed that the population is, on the average, located along the centerline of the plume, the following equation describes the long-term average concentration to which it is exposed:

$$\bar{\chi} = \frac{2Qf}{\sqrt{2\pi}\; u\sigma_z\,(2\pi x/16)}\, exp\left[-\frac{1}{2}\left(\frac{H}{\sigma_z}\right)^2\right] \qquad (3)$$

The frequency function, *f*, is determined by the amount of time the wind was blowing toward the sector, e.g. from a wind rose.

Concentration of a pollutant at a given distance from a source will also depend on atmospheric stability and wind speed. D. B. Turner [8] has described the application of Pasquill stability categories. Since these categories are used in many of the theoretical workups, they are defined in Table A-1. The stability classifications which are determined from STAR programs are ground-level meteorological observations (surface wind speed, cloud cover, ceiling) supplemented by solar elevation data (latitude, time of day, time of year) as inputs. No vertical sounding data are used.

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[8] Turner, D. B., A diffusion model for an urban area, *J. Appl. Meteorol.* 3(1):83-91, February 1964.

Table A-1. *STABILITY CLASSES AND DEFINITIONS**

Pasquill Stability Class	Definition
A	Extremely unstable
B	Unstable
C	Slightly unstable
D	Neutral (day/night)
E	Slightly stable
F,G	Stable to extremely stable

*The STAR program can be run for up to eight stability classes, including a breakdown of the neutral category into day and night divisions.

The modification in Equation (3) which results from the use of direction-specific stability frequencies is that f now becomes a function of stability, direction, and wind speed [$f = f(\theta, S, N)$, where N represents a given wind speed class]. A direction- and stability-specific frequency is the output of the STAR routine. In order to determine the long-term ground-level concentration, the following formula after the work of Turner [4] is used to give a more accurate picture:

$$\chi(x,\theta) = \sum_S \sum_N \left\{ \frac{2\,Qf(\theta,S,N)}{\sqrt{2\pi}\,\sigma_{zs}\,u_N(2\pi x/16)}\, exp\left[-\frac{1}{2}\left(\frac{H_u}{\sigma_{zs}}\right)^2\right]\right\} \tag{4}$$

Thus, at a given distance for a particular wind direction, the long-term concentration is determined by summating each wind speed (N) within each stability (S), and then summating the concentrations for the range of stabilities. Note that σ_z varies with each stability and that the wind speed, u, is determined by the midpoint of the wind speed class which is being summated. The frequency function $f(\theta,S,N)$ is output from the STAR program. (It must be remembered that a northerly wind will expose a population south of the plant.)

When determined for a series of distances and the 16 directions for which STAR data are available, enough data become available to plot long-term concentration isopleths; this was done in the copper smelting, viscose rayon, coal, and steel studies. The Air Quality Display Model was used by EPA to develop theoretical SO_2 concentration isopleths in the vicinity of Rocky Mountain smelters [9]. Results from this report are presented in the chapter on copper smelting. Since elevation relative to the stack has a large effect on ground-level concentration, this variable was included in that study.

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[9] Estimated Impact of Smelting Operations on SO_2 Concentrations in the Rocky Mountain Area, Staff Report, Air Quality Management Branch, OAP, EPA, August 1972.

For our studies in the rugged hill and valley terrain of Appalachia, two different techniques were used to estimate concentration. In the Johnstown study, published research was used to give an indication of pollutant transport patterns in the Conemaugh Valley under very specific meteorologic conditions.

Dispersion of coal dust from Wyoming County coal preparation plants was modeled by using Equation (3) without STAR data. A representative wind speed was selected, stability was assigned as neutral, sector widths were determined by detailed topographic analysis, and the wind direction was assumed to be entirely regulated by the orientation of the river valley, flowing upvalley and downvalley with equal frequency. This simplification of the Turner model provides the epidemiologist with a high estimate of long-term dosage for coal dust in the ambient air.

CAUTIONARY NOTES

As indicated at the outset of this Appendix, the models and equations presented in the theoretical sections of the chapters in the body of the report are subject to a number of qualifying conditions. More of these variables are taken into account in the full models than in the instances when equations were used. However, results from all data manipulations are roughly comparable, so the transport analysis does serve its purpose from an epidemiological standpoint.

Shortcomings in the various estimates are outlined below:

Emission Inventory — Emission inventories are crude at best, especially in view of the long term pollution potentially leading to chronic disease that was being investigated in this study. Little or no information could be obtained on the history of emissions from the plants under study. One method used was the emission factor without any pollution controls as the historical, worst-case assumption. Changes in process, production rate, and location make this a questionable estimate. Reliable data on production history of the industries was very hard to find.

Emission inventories are subject to error. Also, they use constant average emissions which are not reflective of plant outflow, since pollution type and quantity vary significantly on a daily, weekly, and seasonal basis.

Topographic Considerations — Dispersion estimates and models are worked out for a basically flat terrain. Although several sites are on flat terrain, most have confounding topographic features within five miles of the sites. Others are located in mountainous or hill-and-valley terrain. In the chapter on copper smelting, a modified AQDM based on terrain was used. For the rugged valleys of Wyoming County discussed in the coal mining chapter, valley character and orientation were considered to be the sole determinant of wind flows. Where terrain is more or less flat (distant mountain, rolling hills), Equation (4) was applied using available STAR data. The results are correspondingly more reliable.

Climatologic Data — In most cases where STAR data were used, it had to be obtained from observing stations 10 to 200 miles away.

Mixing-Layer Depths — These are difficult to estimate from ground-level observations. Rawinsonde measurements are more appropriate but are not generally available for sites in this study.

Nature of Study — Since the study is one of chronic diseases, we are interested in the long-term levels of pollutants. This is more a problem with the emission source (Q) than with the meteorological data since the latter can be averaged over a multiple-year period and an average taken. In addition, equations defining long-term concentrations are worked out from short-term, point source measurements. Although we do use point sources in this study, the shortcomings in use of these equations for long-term averages must be considered.

SYMBOLS USED

d = inside stack diameter

f = frequency with which wind flows in given direction

$f(\theta,S,N)$ = frequency with which wind flows in given direction; output of STAR routine (relative frequency function)

H = effective stack height

h = physical stack height

Δh = rise of plume above stack

L = height of the stable layer aloft (mixing height)

p = atmospheric pressure

Q = emission rate

S = stability class

σ_y = standard deviation of lateral component of plume

σ_z = standard deviation of vertical component of plume

σ_{zS} = standard deviation of vertical component of plume as function of stability class S

T_a = ambient temperature

T_s = stack gas temperature

u = wind speed

u_N = average wind speed of wind speed class N

v_s = stack gas exit velocity

χ = long-term average concentration

x,y,z = distances in Cartesian coordinate system

Appendix B

COPPER SMELTING INDUSTRY CHARACTERIZATION

Appendix B

COPPER SMELTING INDUSTRY CHARACTERIZATION*

GENERAL DESCRIPTION OF THE INDUSTRY

The primary copper industry includes mining of ore, preparing ore for smelting, smelting, and refining.

Ores

The primary copper industry is characterized by the type of ore being processed. The bulk of most ore deposits are copper sulfides; however, the upper portions of deposits are generally oxides (see Table 1). Gold, silver, nickel, zinc, arsenic, and lead are often associated with copper ore. The average grade of ore presently being mined is 0.7% copper. The process physically separates copper ore from worthless rock, gangue, and chemically separates copper ore from other elements. Different processes are used to separate copper from the two general ore types (oxide and sulfide). See Figure 1.

Table 1

COPPER ORES[2]

Sulfide:	Chalcocite	Cu_2S
	Covellite	CuS
	Chalcopyrite	$CuFeS_2$
	Bornite	Cu_5FeS_4
	Enargite	Cu_3AsS_4
	Tetrahedrite	Cu_3SbS_2
Oxide:	Cuprite	Cu_2O
	Tenorite	CuO
	Malachite	$CuCO_3 \cdot Cu(OH)_2$
	Azurite	$2CuCO_3 \cdot Cu(OH)_2$
	Chalcanthite	$CuSO_4 \cdot 5H_2O$
	Brochantite	$CuSO_4 \cdot 3Cu(OH)_2$

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*This appendix was prepared by Carl B. Bailey.

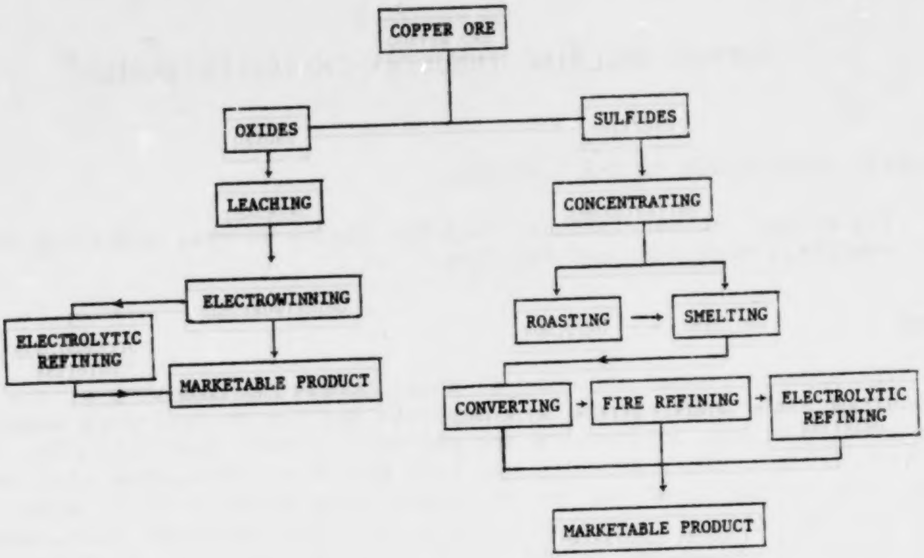


Figure 1. PRIMARY COPPER PROCESSES

Mining

There are two techniques used to mine copper—underground and open pit mining. Ore deposits containing a high percentage of copper and located far below the surface are mined by the underground method. However, most high-grade (high-percentage) ore deposits have been depleted. Low-grade ore, which requires the mining of large quantities of ore (25,000 tons per day) to be profitable, is mined by the open pit method.

Copper Processes

Leaching: Oxide ores are treated with sulfuric acid which dissolves the oxide and leaves the gangue unaffected. The ore is agitated and soaked with the leach solution for several days. The copper is recovered from the solution by combinations of various methods of washing, filtering, settling, or electrowinning.

Electrowinning: The copper, now in the form of copper sulfate, can be recovered from the leach solution by electrolysis. An insoluble anode and copper cathode are placed in the solution creating cells through which an electric current is passed. The copper is chemically released from the sulfate and deposited on the cathode. Copper in this form is 98% pure and is marketed or further refined.

Concentrating: Sulfide ores may be processed by various combinations of the following processes: concentrating, roasting, smelting, converting, and refining. After the mined ore is crushed to a fine size, it is violently agitated with air in a concentrator. Water and reagents in the concentrator create a froth that adheres selectively to the sulfide ore. As a result, the gangue sinks to the bottom of the tank and the ore (20% to 30% Cu) floats to the surface and is removed.

Roasting: The roasting process heats the concentrate in one of two types of roasters—multiple hearth or fluid bed. The copper-sulfur ratio is adjusted for optimum results by removing sulfur as sulfur dioxide after oxidation. Also, volatile material is expelled. The resulting material, calcine, is charged while hot to a reverberatory or electric furnace and smelted.

Smelting: The first part of the smelting process produces a molten material of iron sulfides and copper sulfides, called matte, in a reverberatory (reverb) or electric furnace. In the molten state the matte is heavier than the gangue and they separate. When the gangue is heated, oxides which are called slag are formed. A flux, limestone or silica, is added which aids the separation of slag from the matte. At this point, the slag and matte are removed separately, and the matte enters the converter.

Converting: In the converter, a stream of air is forced through the matte. The iron and sulfur react with oxygen to release large amounts of heat. As air flows through the molten matte, a flux (silica) is added to form an iron-silicate slag, and removes all iron from the matte.

$$\underset{\textit{(matte)}}{2\,CuFeS_2} + \underset{\textit{(air)}}{4O_2} + \underset{\textit{(flux)}}{2\,SiO_2} \rightarrow \underset{\substack{\textit{(sulfur}\\ \textit{dioxide)}}}{3SO_2} + \underset{\textit{(slag)}}{2\,FeSiO_3} + \underset{\substack{\textit{(cuprous}\\ \textit{sulfide)}}}{Cu_2S} + \Delta$$

The slag is removed and air continues to oxidize the sulfur.

$$Cu_2S + O_2 \rightarrow 2\,Cu + SO_2 + \Delta$$

At the end of this reaction, all sulfur is oxidized and copper, some copper oxides, and a few impurities remain. This material is 98.5% copper, called blister copper.

Fire Refining: The blister copper is transferred to a small reverberatory furnace in a molten state or is melted in one. Again, air is forced through the material to oxidize all impurities which rise to the top and are removed. The ends of green logs (poling) or reducing gases are then passed through the material to reduce the copper oxides to copper metal. The molten metal is then cast into slabs (tough-pitch copper) and sold or cast into anodes for electrolytic refining.

Electrolytic Refining: The anodes are placed in cells with an electrolyte (copper sulfate and sulfuric acid) and a pure copper cathode starting sheet; then an electric current passes through the cell. Copper ions migrate from the anode to the electrolyte and are deposited on the cathode. The electrolytically refined copper contains about 99.9% copper.

SITE-SPECIFIC INDUSTRY CHARACTERIZATIONS

The plant processes, age, products, and air pollution controls of the study sites are summarized in Table 2. The Kennecott and Magma plants are relatively new, and the Inspiration plant is the only one in the United States that uses the leaching and electrowinning processes.

EFFLUENTS AND EMISSIONS

Effluents

The plants in the study counties do not have distinct discharges. Because of their geographic location, the sites in Arizona have an acute water problem; consequently, no water is wasted by the industry. However, material from tailings ponds associated with concentrators can leach into the ground. Rain can carry materials into the environment from the mines and plants.

Surface water and rainfall are scarce in Arizona; consequently, water loss at discharges is uneconomical. For instance, in the Miami-Hayden-San Manuel area, the mean relative humidity is less than 40%; the mean annual number of days with a maximum temperature of 90°F and above is greater than 90 days; and the mean annual precipitation is only 18 inches. Process water used for acid plant blowdowns, cooling of anodes, etc., is recycled. Operations that use a water stream to cool and granulate hot slag are not utilized by the study plants. Water is lost mainly through evaporation, and there are no point source discharges.

Tailings pond associated with concentrators may be a source of contamination to ground water resources. Water may leach the tailings if they are soluble. Again, evaporation is probably the greatest method of water loss. Also, the soil permeability will affect the amount of leaching.

The mean annual 18 inches of rain in the study area may carry contaminants to water resources. Rain runoff from the plants may carry plant materials as process chemicals and spills to water resources. However, the annual rainfall does not seem to be a major or continual agent contributing to pollution.

Table 2

STUDY SITE PLANT DESCRIPTIONS[3]

COMPANY/LOCATION	AGE	ELECTROLYTIC REFINING	PRODUCTS	PROCESS DESCRIPTION
ASARCO Hayden, Arizona	1912	No	Blister copper 180,000 t/yr. (Anodes)	• Roasting (multiple hearths) • Smelting • Converting
Kennecott Hayden, Arizona	1958	No	Blister copper 80,000 t/yr. (Anodes)	• Roasting (fluid bed) • Smelting • Converting
Magma San Manuel, Arizona	1955	Yes	Electrolytically Refined 57,000 t/yr. ----------- Fire-Refined 183,000 t/yr.	• Smelting • Converting • Fire Refining • Electrolytic Refining
Inspiration Miami, Arizona	1911	Yes	Electrolytically Refined 75,000 t/yr. --------------- Blister copper 118,000 t/yr.	• Smelting • Converting • Fire Refining ------------- • Smelting (Electric Furnace) • Converting • Fire Refining -------------- • Leaching • Electrowinning
Magma Superior, Arizona	Closed 1971			
Phelps-Dodge Douglas, Arizona	1904	No	Blister copper 4,100 t/yr. -------------- Fire-Refined 142,000 t/yr.	• Roasting (multiple hearth) • Smelting • Converting • Fire Refining
Phelps-Dodge Ajo, Arizona	1917	No	Blister copper 55,400 t/yr.	• Smelting • Converting
Phelps-Dodge Morenci, Arizona	1942	No	Fire-Refined 182,000 t/yr.	• Roasting (fluid bed) • Smelting • Converting • Fire Refining
Anaconda Anaconda, Montana	1903	No	Fire-Refined 202,000 t/yr.	• Drying (multiple hearth) • Smelting • Converting • Fire Refining
Kennecott Magna, Utah	1916	Yes	Fire-Refined 190,000 t/yr.	• Smelting • Converting • Fire Refining

Emissions

There are three major sources of emissions in a copper smelter. Tables 3 and 4 give the general ranges of SO_2 emissions associated with each process. Other miscellaneous sources of emissions are fugitive emissions. Such sources include:[5] hot calcine transfer points, reverb feed and discharge points, converter rollout, and leaks in flues and ducts. Fugitive emissions are difficult to quantify and have been estimated between 0 and 12%[5] of the total amount of SO_2 generated by the smelter. Table 5 lists volatile metals found in domestic copper ore concentrates that may become emissions. Table 6 gives particulate and SO_2 emission estimates for the copper smelters in Arizona. Since size distribution of particles was not available, the unit "tons per day" does not indicate the amount of respirable size (0.3 μ to 5.0 μ aerodynamic diameter) emitted.

Table 3

RANGES OF SO_2 EMISSIONS, PROCESS-SPECIFIC[7]

PROCESS	% TOTAL SO_2 EMISSIONS	
	With Roasting	Without Roasting
Roasting	20-50	---
Smelting	10-30	20-40
Converting	40-50	60-80

Table 4

EMISSION FACTORS[a] FOR PRIMARY COPPER SMELTERS WITHOUT CONTROLS[6]

TYPE OF OPERATION	PARTICULATES[b]		SULFUR OXIDES	
	lb/ton	kg/Mt	lb/ton	kg/Mt
Roasting	45	22.5	60	30
Smelting (Reverberatory)	20	10	320	160
Converting	60	30	879	435
Refining	10	5	-	-
Total Uncontrolled	135	67.5	1250	625

[a] Emission factors expressed as units per unit weight of concentrated ore produced.

[b] Electrostatic precipitators have been reported to reduce emissions by 99.7 percent.

Table 5

TYPICAL LEVELS OF VOLATILE METALS IN DOMESTIC COPPER ORE CONCENTRATES[7]

	CONCENTRATION LEVEL	% CONCENTRATES SURVEYED
LEAD	<5000 ppm	96%
	5000 - 20,000 ppm	2%
	≥ 20,000 ppm	2%
ZINC	10,000 ppm	67%
	10,000 - 40,000	32%
	≥40,000	1%
ARSENIC	200 - 1000 ppm	88%
	1000 - 10,000 ppm	10%
	>10,000	2%
CADMIUM	<1000 ppm	100%
BERYLLIUM	<10 ppm	100%
VANADIUM	<100 ppm	100%
ANTIMONY	<200 ppm	97%
	>200 - 5000 ppm	3%
	>5000 ppm	<1/2%
TIN	<1000 ppm	100%

Table 6. STACK DATA AND THEORETICAL EMISSIONS[7]

PLANT	PROCESS	NO. OF STACKS	HEIGHT FT.	FLOW RATE $x10^3$SCFM	PARTICULATES T/D	SO_2 T/D	SO_2 CONC. PPM	MISCELLANEOUS
ASARCO	Roasting-Smelting	1	1,000[2]	403	1.5	290	5900	
	Converting	1	30	81.2	∼0	21.8	2200	
Kennecott	Roasting (50% Converting)	1	100	75	∼0	12	1300	
	Smelting (50% Converting)	1	600	211	1.1	187	7250	
Magma	Smelting	1	515	175	1.79	313	15000 20000[3]	15000[3] H_2O 83000[3] Inert Natural Air Components
	Converting	1	550	117	.54	767	54000	
	Converting[3] (Acid Plant)	2	250			30	2160	
Inspiration	Smelting	1	275	260.8		78	2500	
	Converting	1	200	163.4		296	15200	
Phelps-Dodge Douglas	Roasting-Smelting	1	544	470	39	902	15700	
	Converting	1	556	264	9	492	15200	
Phelps-Dodge Morenci	Roasting-Smelting	1	600	310	14.7	526	14000	
	Converting	1	600	406	0.8	768	15400	
Phelps-Dodge Ajo	Smelting-Converting	1	360	397	0.7	380	7000	
Anaconda	Drying-Smelting-Converting	1	925	1,950	22.5	790	3300	

- Control of emissions has been practiced only in recent years, and then with various degrees of efficiency. In general, many smelters use balloon flues and cyclones for particulate removal; however, these devices are very inefficient in removing respirable size particles, as shown in Figure 2. Electrostatic precipitators (ESP), on the other hand, are capable of removing respirable particles (Figure 2). For the removal of SO_2, many problems exist and are well documented in the literature. Proposed systems for the primary copper industry are:[7]

  - Sulfuric Acid Plants
  - Elemental Sulfur Plants
  - Calcium-Based Scrubbing Systems
  - Dimethylaniline (DMA) Scrubbing
  - Ammonia Scrubbing
  - Sodium Sulfite-Bisulfite Scrubbing

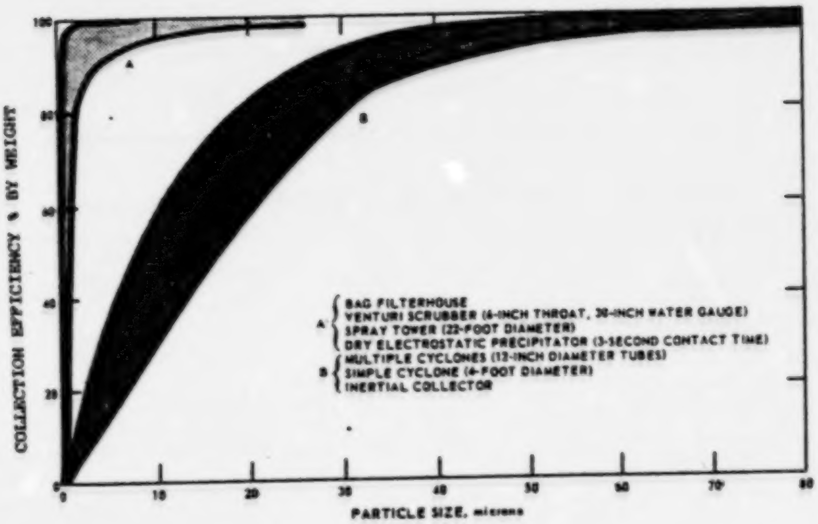


Figure 2

COMPOSITE GRADE (FRACTIONAL) EFFICIENCY CURVES BASED ON TEST SILICA DUST[7]

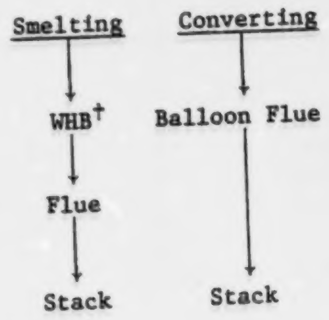
Often these processes require a constant, uniform gas stream; consequently, the plant operation procedures may be modified. For example:[5]

- A minimum and maximum number of converters blowing at one time.
- Converters finish flowing one at a time.

In addition, collection devices are needed around emission sources in the plant to reduce fugitive emissions, which would otherwise not be treated.

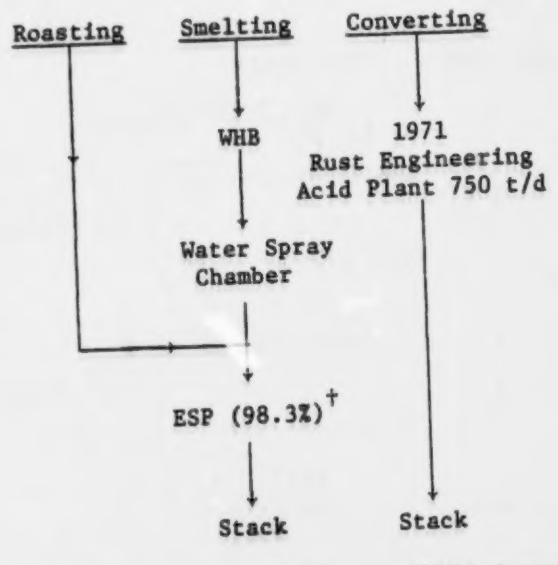
In general, emissions were not controlled until the 1970's. Now, particulates are usually well controlled and SO_2 is poorly controlled, if at all. The theory that "the solution to pollution is dilution" is still practiced—ASARCO increased their converter stack height from 300 to 1,000 feet.

Figures 3 through 10 are schematic respresentations of site-specific emission control devices.



†WHB = Waste Heat Boiler

Figure 3. INSPIRATION EMISSION CONTROL DEVICES



†ESP = Electrostatic Precipitator (Efficiency)

Figure 4. ASARCO EMISSION CONTROL DEVICES[7]

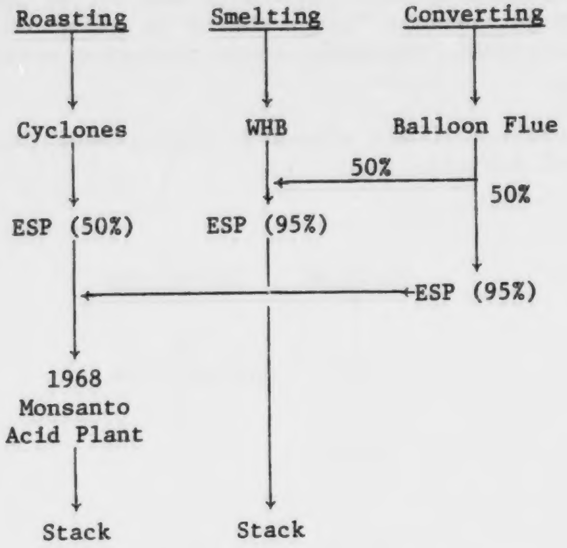


Figure 5. KENNECOTT EMISSION CONTROL DEVICES[7]

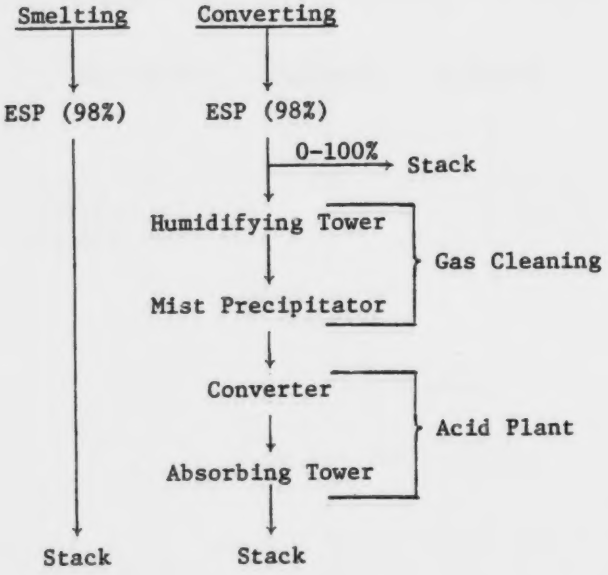


Figure 6. MAGMA EMISSION CONTROL DEVICES[5]

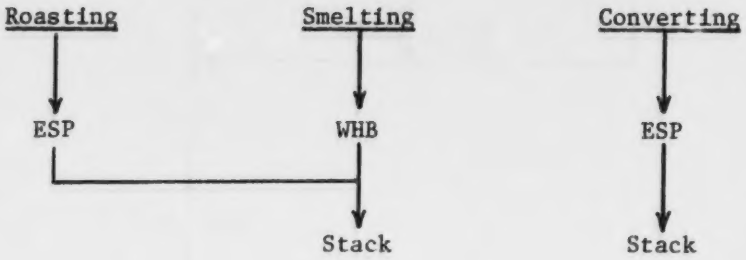


Figure 7. PHELPS-DODGE/DOUGLAS EMISSION CONTROL DEVICES

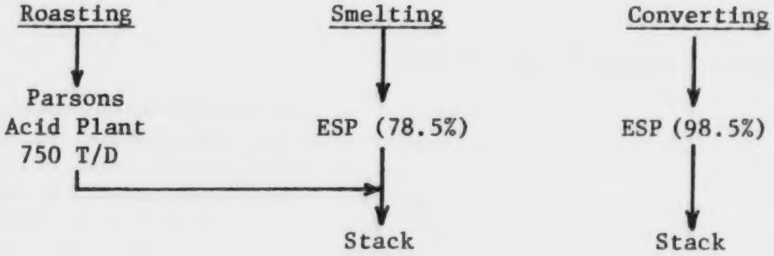


Figure 8. PHELPS-DODGE/MORENCI EMISSION CONTROL DEVICES

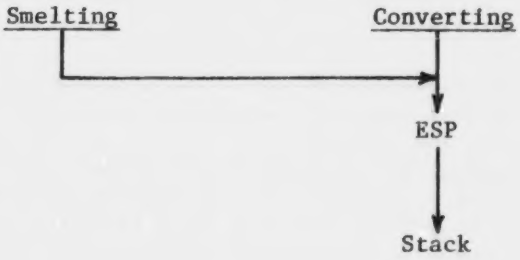


Figure 9. PHELPS-DODGE/AJO EMISSION CONTROL DEVICES

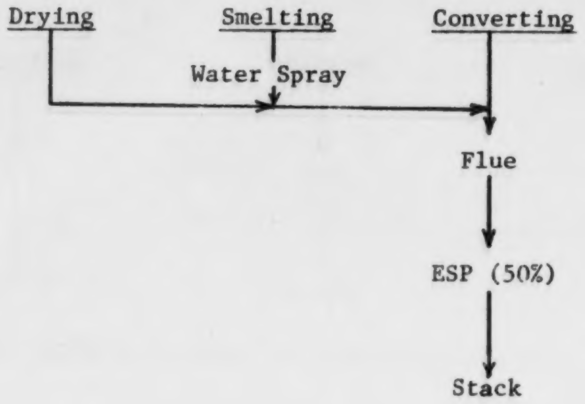


Figure 10. ANACONDA EMISSION CONTROL DEVICES

MAGMA: PROCESS DESCRIPTION & HISTORY[5]

The Magma Copper Company operates an underground mine, concentrator, smelter, electrolytic refinery, and continuous rod-casting operation in San Manuel, Arizona. Smelting of copper concentrates began in 1956 and has had two expansions—in 1965 and 1971. Ore production increased from 30,000 to 40,000 tons per day (t/d) and then to 62,500 t/d, respectively. In 1975 Magma employed 4,151 people and produced 187,427 tons of copper.

The San Manuel Mine ore deposit is chiefly chalcopyrite with a copper content of about 0.75%. Metallic minerals include pyrite, chalcopyrite, chalcocite, molygdenite, silver, and gold. The economically feasible (from a mining standpoint) deposit was initially fishhook-shaped and up to 400 feet thick. The average depth of overburden was 670 feet. The daily capacity is 62,500 tons, and block caving is the mining technique employed.

A train carries the ore from the mine to the plant six miles away. The train of 36 100-ton-capacity cars makes 18 round trips daily.

Crushing, grinding, and flotation concentrate the ore. Primary, secondary, and tertiary crushing reduce the ore to less than an inch in size. Grinding in rod mills and then in ball mills precedes flotation. Retention time in the flotation cells (1,056 cells of 40-cubic-foot capacity, and 63 cells of 300-cubic-foot capacity) is nine minutes. Concentrate from the 300-cubic-foot cells is reground in the ball mill and recirculated to the 40-cubic-foot cells. Molybdenum can be recovered by flotation in another series of cells. The gangue from the cells is now called tailings and is thickened and discharged to tailings ponds.

The copper concentrate is fed to three reverb furnaces (102 feet long). The concentrate is about 28% copper and 10% moisture. The side-feed reverbs are oil-fired or natural gas-fired. Smelting occurs at about 2700°F and combustion air is preheated. Slag is dumped close to the plant. The matte is 32% to 35% copper.

The matte is transported to one of six Pierce-Smith type converters by 300 cubic feet of ladles. An 18-hour period is required to produce blister copper. Slag is returned to the reverbs.

Ladles again transport the molten copper to four holding furnaces for fire refining, 99.7% copper. Anodes weighing 820 pounds are cast and cooled by water sprays.

The electrolytic refinery came on line in 1971. Anodes are suspended in refining cells, 4 inches apart, with starter sheets between the anodes. Electric current, at 21,000 amps per cell and DC volts at 0.23 per cell, provide a current density of 24 amps per square foot. After 28 days, the 820-lb anode is 130 pounds and recycled to the converter. Cathodes are 345 pounds.

The electrolyte circulates through cells at a rate of 4 to 5 grams per minute. It contains 48 g/ℓ copper and 185 g/ℓ sulfuric acid, and the temperature is controlled at 150°F. Copper in solution is removed in an electrolytic purification (liberator) section which has an insoluble lead anode and starter cathodes. Copper ions leave solution and desposit on a low-quality liberator cathode which is recycled to the smelter. Impurities, which settle out into the slime, are collected, acid-leached, filtered, and dried. This material is then shipped to a precious metal refinery for recovery of gold, silver, and selenium. The slime is washed out of the refining cells every 28 days. Some cathodes are melted and cast into 5/16" and 13/32" diameter rods.

Emission Controls

To date (1 July 1977), Magma is the only company to provide any information after being contacted. Figure 6 gives a description of their emissions by a comprehensive plant operation program and ambient monitoring system. Converter operations are synchronized to provide a constant, uniform offgas to the acid plant for greater SO_2 efficiency. In addition, their eight ambient air quality monitoring stations' data are analyzed with meteorology reports. Consequently, guidance information about possible adverse weather conditions resulting in air pollution episodes can be anticipated and plant operations may be curtailed.

INSPIRATION CONSOLIDATED COPPER COMPANY: HISTORY[1,8,9]

1916 — First production of copper from the underground mine and flotation process.

1926 — Began production of ferric-sulfate leaching of mixed oxide-sulfide ore and of ectrolytic copper.

1933 — By May, all mining and smelting stopped due to the depression.

1935 — Production began again, in September.

1948 — Production from open-pit mining began and the leaching plant was enlarged.

1954 — Underground mining was stopped and completely replaced by pit mining.

1958 — Molybdenum recovery began.

1960 — The International Smelting and Refining Smelter was purchased and a new refinery built.

1962 — The "Christmas" underground mine began to produce.

1966 — Treated ore production increased from 16,500 to 20,000 t/d.

1968 — Capacity increased to 25,000 t/d of ore and the Christmas mine was converted to an open pit, and the "Ox-Hide" mine was developed. Copper fabrication and rod plant.

1972 — Pollution control construction began—electric copper furnace and acid plant.

ANACONDA PROCESS DESCRIPTION[11]

The Anaconda Company smelting complex at Anaconda, Montana, consists of two plants—the pyrometallurgical smelter and the hydrometallurgical smelter. The two plants can produce 35 million and 6 million pounds of copper per month; however, legal agreements limit the combined output to 35 million pounds per month.

Major processes at this complex consist of four reverberatory furnaces, the electric furnace, six converters, two refining furnaces, a baghouse, an electrostatic precipitator, a sulfuric acid plant, two lime kilns, a boiler house, the Arbiter plant, concentrate drying and blending area, tailings ponds, and slag pile.

The Anaconda Company currently has variances for particulate emissions from the main stack and for the 90% SO_2 control rule which applied to the smelter. Compliance with both of these rules is related to completion of new smelting facilities followed by expansion of the sulfuric acid plant. Compliance with the particulate emission rule will be achieved for the main stack and matte tapping hoods when the reverberatory furnaces are shut down (probably in 1975). Compliance with an interim 75% control rule will be achieved in early 1977, followed by a 6-month review of the applicable standards.

HISTORY OF THE ANACONDA SMELTING OPERATION[11]

1883 — Smelter construction started. A concentrator building was completed.

1883 — Upper works built.

1884 — Smelter construction was completed. It has a capacity of 10 million pounds per month. The refinery had a capacity of 30 million pounds per month and another of equal capacity was started. Custom smelting was done from the start.

1887 — The Great Northern Railroad was extended to Butte so that ores and copper could be shipped to Great Falls.

1887-1888 — Lower works built when the upper works were found to be too small.

1889 — The lower works burned in March. They were rebuilt before winter with a larger capacity. In 1892 the smelter output was 100 million pounds of the metal.

1894 — On January 1, the Butte, Anaconda and Pacific Railroad went into operation.

1896 — Sulfuric acid manufacture from the flue gases started. Leaching of tailings was carried on successfully. Electrolytic refining of copper started.

1896 — The company sold 107 million pounds of copper, 4.5 million ounces of silver, and 14,400 ounces of gold.

1898 — Half of the company's yearly output of 124 million pounds (10.03×10^6 lb/mo) of copper was electrolytically refined.

1899 — Amalgamated bought Anaconda Copper Mining Co.

1902 — The Washoe Smelter was first used on January 25. Included in the facilities was the original tall stack and settling chamber.

1903 — The Anaconda Copper Mining Co. paid $330,000 in damages to local ranchers for animal loss.

1903 — A 300-foot stack and the existing settling chamber were installed.

1910 — Mining options were first obtained in Chile by an American firm.

1914 — Electrolytic refining of zinc was developed.

1918 — The present main stack and electrostatic precipitators were installed to remove solid and liquid matter from the flue gas.

1919 — The main stack was first used.

1919 — The company constructed a plant at the reduction works for production of high-quality fertilizer.

1923 — The Anaconda Co. bought the Chile Copper Co. which had started operations at Chuquicamata, Chile, in 1915.

1931 — Use of natural gas at the smelter was started. All coal burning apparatus was replaced with gas burners.

1941 — The manganese concentrator and nodulizing plants were completed. This was at the request of the U.S. Government to alleviate a shortage created by World War II.

1955 — The Anaconda Reduction Department produced 252.3 million pounds of copper, 153.6 million pounds of zinc, 145,300 tons of sulfuric acid, 89,500 tons of fertilizer, 73,800 tons of manganes nodules, and large quantities of arsenic.

1958 — An ammonium phosphate plant was put into operation.

1959 — The zinc crusher, zinc concentrator, and manganese flotation plant were closed.

1961 — The phosphate plant was shut down.

1964 — The arsenic plant was shut down.

1966 — A fifth converter was added.

1967 — The Montana Legislature enacted the "Clean Air Act of Montana."

1969 — The zinc leaching and purification plants were closed.

1969 — Ambient air standards governing sulfur oxides at smelters, regulation 90-015, was passed by the Montana State Board of Health in November.

1970 — On April 30, 1970, regulation 90-008 was adopted. It became effective on June 30, 1973.

1971 — A Johnson-Marsh Chem-Jet Dust Suppression system was installed at the East Anaconda Crusher.

1971 — The new flue system removing off-gases from the reverberatory furnaces and converters was completed.

1971 — Anaconda's Chilean holdings were nationalized.

1971 — On November 17, the Anaconda Co. was issued a construction permit for "roasters, smelting furnaces, and converters" to be located at Anaconda, Montana.

1971 — On December 15, the Anaconda Company's request for a change in the State regulations concerning sulfur oxide emissions from primary non-ferrous smelters was denied.

1972 — In January, Governor Anderson submitted the "Implementation Plan for Control of Air Pollution in Montana" to the Environmental Protection Agency without the section on SO_2 control.

1972 — The zinc refining operations at Great Falls ended.

1972 — On July 27, EPA proposed a regulation to control the emission of sulfur oxides at the Anaconda Company smelter and to hold public hearings on the proposed regulation 30 days later.

1972 — On July 28, a construction permit for "water-cooled converter hoods, converter uptakes and flues, gas scrubber, fiberglass flue to acid plant, 660 TPD acid plant with storage and loadout facilities" was approved.

1972 — On August 30, EPA held a public hearing in Helena pursuant to proposed regulations for control of sulfur oxide emissions from smelters.

1972 — On September 26, the Anaconda Company filed a civil suit against William D. Ruckelshaus (EPA Administrator) and others, asking that EPA be required to hold an adjudicative hearing and prepare an Environmental Impact Statement prior to promulgating rules for control of sulfur oxide emissions from smelters.

1972 — On December 19, the federal district court in Denver rendered its decision in favor of the Anaconda Company in its suit against EPA (see 1972, September 26).

1973 — On February 8, Notices of Violation and Order To Take Corrective Action were issued by the Air Quality Bureau for excessive visible emissions from the two lime kiln stacks and the foundry cupola furnace. Both lime kiln emissions were corrected by June 1. Approval of control equipment for the foundry has been approved. Installation is expected shortly.

1973 — On February 12, the Anaconda Company requested a variance from regulation 16-2.14(1)-S1470(2), formerly 90-008.

1973 — Use of the new water-cooled hoods and flue system was started.

1973 — The seventh and eighth converters were added.

1973 — Operation of the 660 t/d sulfuric acid plant was started in June.

1973 — In June, the U.S. Supreme Court upheld rulings by lower courts in a lawsuit filed by the Sierra Club against EPA that EPA must disapprove all State implementation plans which did not contain provisions to prevent degradation of existing air quality in areas that had cleaner air than allowed by the national standards.

1973 — In July, the Anaconda Company requested and was granted a variance from regulation 90-004 governing particulate emissions from the smelter. This variance expires in July 1974.

1973 — In July, the Anaconda Company started construction of a baghouse to remove particulate matter from the main flue gases. Expected completion date is August 1, 1974. This baghouse will not treat all gases going to the main stack until the new electric furnace replaces all but one of the reverberatory furnaces and the flue-solids roaster is operational.

1973 — In July, the U.S. Court of Appeals, Tenth Circuit, ruled that the federal district court was not the proper one to hear the case of the Anaconda Company vs. William Ruckelshaus et al. (see September 26 and December 19, 1972), that the impact statement was not necessary and that EPA had acted in a proper manner in establishing rules for sulfur oxide controls.

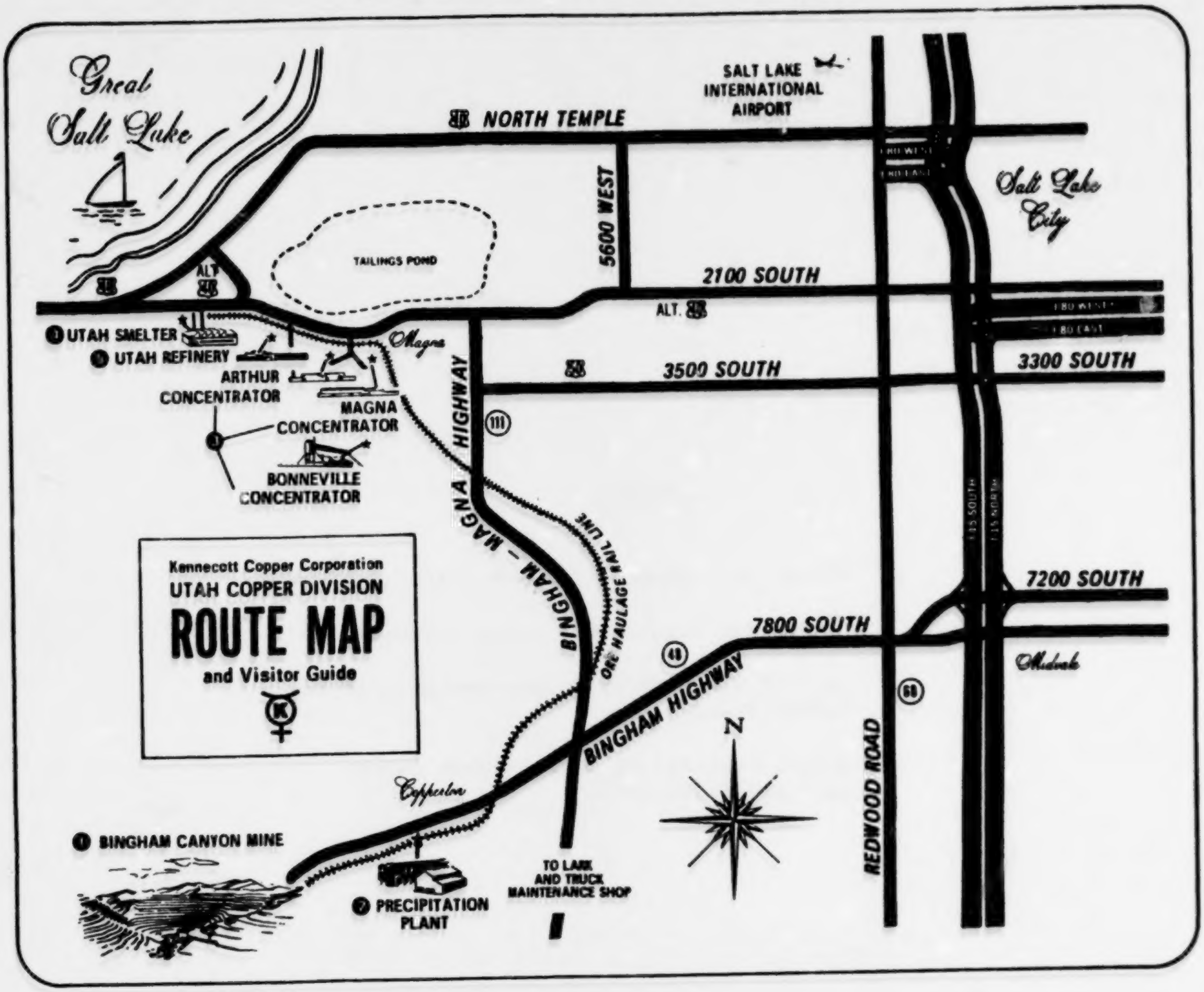
1973 — On July 26, the Anaconda Company initiated action to classify certain new equipment at their plant as air pollution control equipment and, as such, be eligible for a class seven taxation rate.

1973 — In September, EPA held one of four scheduled public hearings (in Denver) to try to determine the best of four possible approaches to achieving "no significant deterioration" of air quality.

ADDENDA

- Map of the Kennecott copper works at Magna
- Production figures for Magna (1940-1971)
- List of all primary copper smelters in the United States
- Annual capacity of four Arizona copper smelters (1927-1973)

KENNECOTT COPPER COMPANY, MAGNA, UTAH



The copper production cycle starts at the Bingham Canyon Mine ❶, where 108,500 tons of ore and over 300,000 tons of overburden, or waste material, are removed daily. Because overburden contains trace amounts of copper, it is treated with a solution which is subsequently processed at the precipitation plant ❼. Ore hauled by train from the mine is processed through three concentrator plants ❸ where it is crushed, ground and concentrated to nearly 30 percent copper. The smelter ❹ takes the concentrate and produces blister copper, 98.6 percent pure; then copper anodes 99.5 percent pure. At the electrolytic refinery ❺ the copper is further purified to 99.96 percent and cast into various shapes for Kennecott customers.

Furnished by:

Communications Department
Utah Copper Division
Kennecott Copper Corporation
P. O. Box 11299
Salt Lake City, Utah 84111

ANNUAL PRODUCTION (10^3 tons) OF COPPER BY THE SMELTER AT MAGNA, UTAH, 1940-1971

Year	Copper Production	Year	Copper Production
1971	260	1955	233
1970	296	1954	212
1969	297	1953	269
1968	228	1952	283
1967	169	1951	271
1966	265	1950	279
1965	259	1949	197
1964	200	1948	227
1963	203	1947	267
1962	218	1946	114
1961	213	1945	226
1960	219	1944	302
1959	145	1943	326
1958	189	1942	318
1957	238	1941	271
1956	251	1940	249

Source: Health Consequences of Sulfur Oxides: A Report from CHESS. 1970-1971. U.S. EPA, Office of R&D, National Environmental Research Center, Research Triangle Park, N.C.

PRIMARY COPPER SMELTERS IN THE UNITED STATES

- American Smelting and Refining Company
    - El Paso, Texas
    - Hayden, Arizona
    - Tacoma, Washington

- The Anaconda Company
    - Anaconda, Montana

- Cities Service Corporation
    - Copperhill, Tennessee

- Inspiration Consolidated Copper Company
    - Miami, Arizona

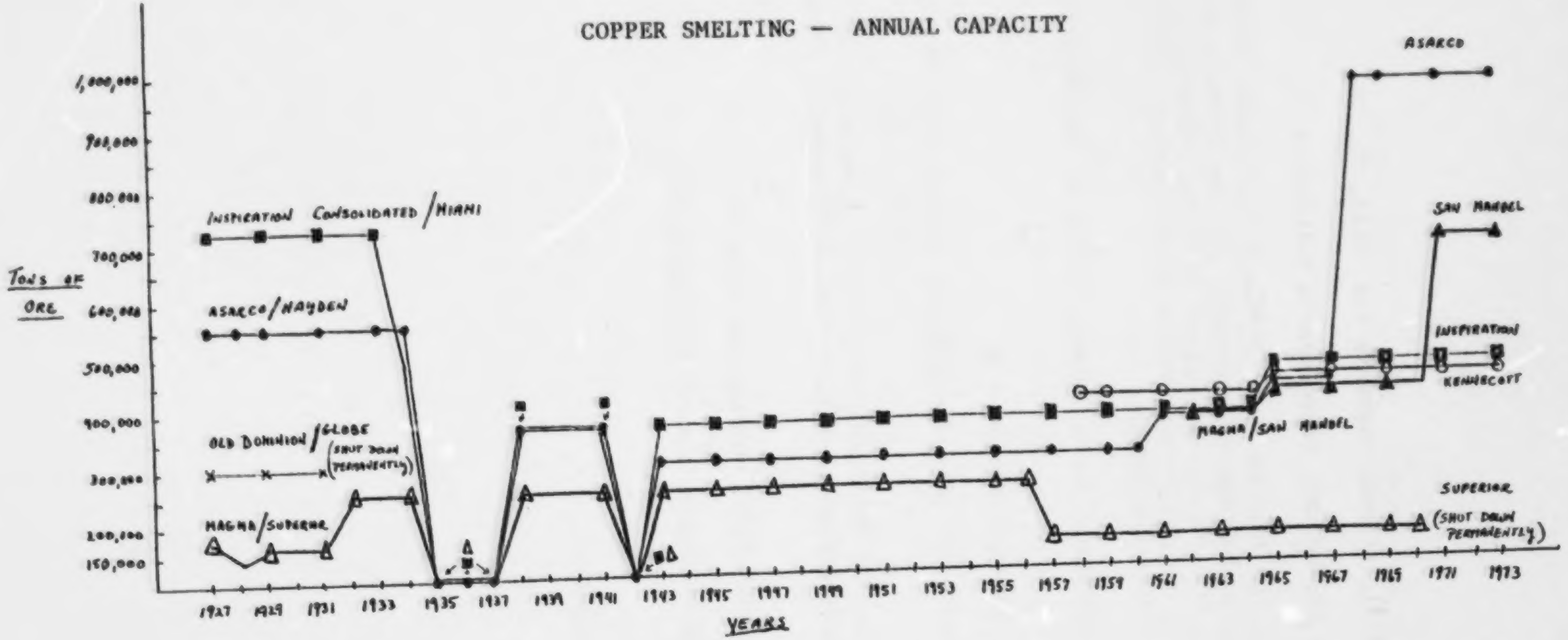
- Kennecott Copper Corporation
    - Garfield, Utah
    - Hayden, Arizona
    - Hurley, New Mexico
    - McGill, Nevada

- Magma Copper Company
    - San Manuel, Arizona

- Phelps-Dodge Corporation
    - Ajo, Arizona
    - Douglas, Arizona
    - Morenci, Arizona

- White Pine Copper Company
    - White Pine, Michigan

COPPER SMELTING — ANNUAL CAPACITY



SOURCE: American Bureau of Health Statistics, Yearbook.

NOTE: Magna Co. Smelter at San Manual started production in 1962, ▲-▲-▲; Kennecott started in 1958, -o-o-o . All of the data are based on the capacities of the smelter reverbatories, except for the data for the Old Dominion Smelter, which had no reverbatory. Data for the Old Dominion is based on blast furnace capacity.

The capacity of copper smelting works is given as estimated by the respective proprietors, stated in tonnage of capacity for smelting materials that yield a product. Capacity in terms of copper product varies with the grade of the ore charged. Ore and concentrates are metallurgically synonymous terms.

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11. Montana Air Pollution Bureau.

Appendix C1

ETIOLOGY OF DISEASES ASSOCIATED WITH COPPER SMELTING

Appendix C2

SELECTED ANNOTATED BIBLIOGRAPHY OF LITERATURE RELATING DIGESTIVE DISEASES AND COPPER SMELTING

Appendix C1

ETIOLOGY OF DISEASES ASSOCIATED WITH COPPER SMELTING*

COPPER SMELTING AS A SOURCE OF TOXIC POLLUTANTS

Studies of many mining operations or heavy industry have identified diseases that are directly correlated with the primary product itself, i.e., coal mining and coal miners' pneumoconiosis or fluorescent lights and beryllium pneumonitis. Copper smelting plants, on the other hand, emit a large number of trace and heavy metals, many of which are necessary in living organisms. Their mere presence in slightly elevated levels with certain diseases does not necessarily make a case for disease causation. One of the difficulties with copper smelters is the extreme variety of materials given off, much of which in gross excess may be associated with disease. Almost no long-term, low-dose animal experiments have been done to anticipate the types of disease to expect. Most assumptions of the environment's impact are extrapolated from *high*-dose, long-/short-term exposures; these data derive from workers in mines and plants.

IMPORTANT VARIABLES IN MONITORING COPPER SMELTER DISEASES

Unlike coal mines, copper smelters are unique for several reasons:

1) Some of the trace metals given off are necessary to human and animal biologic functions; therefore, slight aberrations may not be detectable for years or, in fact, generations. Since effects are often delayed and may manifest themselves after exposure has ceased, studies must be conducted on stable populations (1,26).

2) The scope of investigation will of necessity need to be much larger if a serious evaluation of a copper smelters' effect on the environment is to be appreciated. Because of materials released, more subtle diseases may become manifest (vide infra) and need to be monitored (17).

3) Not only should diseases of excess be considered, but also those of deficiencies. With multiple trace metals in competition for binding sites in the gastrointestinal system, absorption of necessary elements may be eliminated leading to decreasing levels of these elements. Pollutants from smelters may be acting like fertilizers that have already modified the mineral content of the soil and its products, creating a manganese-deficiency in grazing animals and a copper-deficiency in grains and plants (27).

4) If a "specific industry-disease association" is to be made, it will be necessary to monitor more extensively the food sources. Arsenic, which is produced by smelters, is concentrated by shellfish but is also

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*This appendix was prepared by Tee L. Guidotti, M.D., in collaboration with Philip Smith, M.D.

used in insecticides extensively and can be found in apple cider and wine. Therefore, the real source of arsenic contamination may be missed if only samples of water and air are monitored. Furthermore, a report from Hopkins soon to be released will report that one of the largest sources of copper in water is copper pipes in common households.

5) All previous studies involving copper smelters, including the *International Journal of Cancer* report on 839 copper smelters, pay only lip service to smoking but do not control for this variable in evaluating lung cancer. In this particular report 10 out of 11 subjects smoked, which is an inordinately high percentage and would in itself be associated with increased incidence of lung cancer. Of further importance is the known exacerbation of smoking and the inhalation of toxic fumes--the cigarette acting as the catalyst (1).

6) Arsenic and possibly other pollutants in excess can lead to paresthesia, muscle weakness, and other types of neurologic deficits. Although many neurologic signs and symptoms are subtle and tend to be overlooked, they often serve as an early marker for toxic effects (16). Manganese and lead toxicity can produce acute self limited psychiatric disturbances. In the case of manganese intoxication, the syndrome very closely resembles schizophrenia; on the other hand, manganese deficiency may produce a Parkinson-like state. It has been postulated that manganese and possibly other trace metals may modulate the dopaminergic apparatus of the brain (17).

7) As has been mentioned, when evaluating the respiratory system it cannot be overemphasized that the impact of smoking (vide supra) must be carefully controlled. When looking at data from a smelter community, not only must the type of smoking be recorded (cigar/pipe/cigarette/marijuana) but so too the duration and sequence of smoking. Sequence refers to whether cigarette smoking occurred before the institution of cigar smoking. This is important because individuals who smoke cigarettes first tend to inhale pipe smoke and, therefore, may have as much lung disease as cigarette smokers. Many medical record departments do not consider pipe smokers.

8) Because manganese, copper, zinc, and selenium are needed for enzyme systems and hormonal balance, diseases of the endocrinologic system must be considered. Deficiencies in manganese may cause sterility in males. Copper is used in intrauterine devices to prevent conception, and in rats copper deficiency causes infertility. There is also some modulation of the adrenal coricosteroids by copper and zinc, although no direct link has been made to specific diseases such as Cushing's or Addison's diseases (17).

9) The teratologic potential of trace metals in man has not yet been investigated. Manganese-deficient chicks have offspring with chonodrodystrophy; copper-deficient lambs have offspring with enzootic ataxia (neurological dyscoordination). Zinc-deficient rats have multiple congenital anomalies and malformations involving the face and limbs. There-

fore, when surveying the smelter community, careful obstetrical histories must be sought. This should include any spontaneous abortions and pathology of aborted fetuses.

10) Acute infections are accompanied by well-documented changes in serum concentrations of iron, zinc, and copper. However, serum concentrations during illnesses do not necessarily reflect body stores; whether or not these elements are necessary for host defense is unproven.

11) Dental abnormalities are often signs of high-dose exposure to sulfuric acid and other acids in aerosolized form. These changes are initially reversible, but with chronic high-dose exposure lead to permanent damage (14).

TOXICOLOGY OF DISEASES OF COPPER SMELTER WORKERS

There are no specific diseases per se of copper smelter workers. As has been mentioned, the variety of pollutants is extensive although the amounts are generally small. The following represent the most likely disease associations that one might anticipate, i.e., pollutants that are both present in higher concentrations and have the best scientific evidence of disease association.

Cadmium

There is good evidence that cadmium is correlated with hypertension. Whether cadmium causes hypertension in humans is controversial, but animal studies do demonstrate an increased incidence of hypertension with long-term, low-dose exposure. In a recent report on 27 copper smelter workers, an increased incidence of proteinuria and renal stones was reported. Cadmium as an air pollutant may be a hazard, especially to a select group—those with alpha-1 antitrypsin deficiency. It is the only trace metal known to decrease alpha-1 antitrypsin and its activity in human serum, thus predisposing the individual to emphysema (18,19).

Arsenic

The increased incidence of skin and lung cancer is well documented, both epidemiologically and in the laboratory; arsenic also affects the neurologic system of man and animal with resultant optic neuritis, parasthesia, neuralgia, and hyperesthesia. There is also the development of "mees lines" in fingernails. The skin often erupts in acute high-dose ingestion leading to erythematous lesions, hyperpigmentation, and keratosis (15). By far the most worrisome association is that of lung cancer in copper smelter workers. This has been attributed to the inorganic arsenic released in the air (1,3,26,28). Because of the unusually high incidence of poorly differentiated epidermoid bronchogenic carcinoma, Newman has suggested that this excess may serve as a marker (25).

Sulfuric Acid/Sulfur Dioxide

Sulfuric acid and sulfur dioxide are both toxic respiratory pollutants, the former perhaps more so than the latter. In one of the best long-term low-dose experiments in dogs, SO_2 and other auto emission products were studied (22). SO_2 alone caused not only pathologic changes in the lung (emphysema) but also early changes in pulmonary function. Amdur has reported the respiratory effects in guinea pigs of both sulfur dioxide and sulfuric acid in mist form. In combination, the pollutants decreased growth and damaged the lung more than either of the two did alone (8).

Lead

The toxic effects of lead have been well documented (13), and low-dose exposure is most likely to result in the following: 1) peripheral neuropathies such as paresthesia and hyperesthesia, also possible are subtle mental disorders such as depression; 2) nephropathy—manifested as an interstitial nephritis; 3) anemia; and 4) gastrointestinal symptomatology—abdominal colic, constipation, and weight loss. Even though toxic symptoms are not usually present until blood levels reach 80 μg/100 mℓ, occupational exposure that results in absorption of 15 g daily can achieve a positive lead balance. Therefore, lead levels can be misleading and at present are based on high-level industrial exposure (16). Cooper reported in 1974 on 7,032 lead smelter and battery plant workers, who were in some cases exposed to high doses of lead, documenting no differences in standardized mortality ratio (SMR) for all causes or life expectancy (24).

Nickel

High exposure to nickel has three relatively distinct hazards associated with it: 1) contact dermatitis that develops when exposed to solutions of nickel; 2) hemorrhagic pneumonia when nickel carbonyl is inhaled; and 3) cancer of the upper respiratory tract (nasal sinuses) and lung if nickel dust is inhaled. Since arsenic is often mixed in, it is unclear which is more at fault. As with other trace metals, there are no long-term low-dose experiments with nickel. There is evidence that nickel in the water ($NiSO_4$) is more readily absorbed than is nickel bound to food.

SUMMARY

Copper smelters release toxic agents but also trace elements essential for biologic function. To evaluate the impact of a smelter on community health, the following should be considered:

- Monitoring of as many food sources as possible, to avoid missing other significant sources of pollutants.
- In-depth evaluation of human systems beyond respiratory, cardiac, and gastrointestinal systems to include endocrinologic, neurologic, and renal systems.

- Teratology and abortion histories of females.
- As detailed a smoking history as possible.

The following represent a list of the diseases most suspect for community risk:

- respiratory—laryngitis, asthma, bronchitis, emphysema, fibrosis, pneumonia
- renal—hypertension, proteinuria, stones
- neurologic—paresthesia, hyperesthesia, neuralgia, paralysis, mental disorders (depression)
- gastrointestinal—granulomas in the liver
- oncologic—cancer of lung and upper respiratory tract (nasopharynx), skin, gastrointestinal tract; leukemia
- endocrinologic—infertility
- dental etching of enamel, caries
- skin hyperpigmentation, keratosis, dermatitis
- reproductive—birth defects, abortions

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Appendix C2

SELECTED ANNOTATED BIBLIOGRAPHY OF LITERATURE RELATING DIGESTIVE DISEASES AND COPPER SMELTING

Copper and Brass Research Association. Copper and health. *6483*:11-20, 1948.

After an extensive historical review of the occupational hazards of working with copper, the review concludes, "Today, so far as we know, copper is negligible as an industrial poison and the ill-health that is found among copper miners or workers with the metal is caused by something else connected with their work—heat or overexertion, or irritating dust, or the presence of lead or arsenic mixed with the copper." [p. 20]

McCaull, Julian. Building a shorter life. *Environment 13*(7):2-15, 38-41, 1971.

Cadmium pollution of the environment is reviewed with respect to basic characteristics, emission sources, uses, concentration levels, and effects on human health. Cadmium dust, fumes, and mist are emitted during the refining of zinc, copper, and lead, as well as during extraction of cadmium. Cadmium is correlated with hypertension, liver damage, bone disease, emphysema in industrial workers, cancer, and kidney impairment.

Fulkerson, S. *Cadmium—the dissipated element—revisited*, Environmental Aspects of Trace Contaminants Program and Atomic Energy Commission Interagency Agreement AG 389. Oak Ridge National Laboratory, Oak Ridge, Tennessee, Energy Division of National Science Foundation Research Applied to National Needs, 1975, 28 p.

Cadmium air pollution is a serious concern near nonferrous metal smelters, especially zinc and lead smelters. Emissions from these operations can lead to soil and water contamination. Emissions from steel mills, incinerators, or coal burning plants are probably insignificant sources of air pollution, or can be reduced to insignificance by proper abatement practices. Estimates of the rate of accumulation of cadmium in the kidney and of the critical concentration (200 ppm) in the renal cortex necessary to cause renal tubular dysfunction have been used to calculate the maximum permissible chronic exposure by various environmental pathways. Cigarettes are a more important source of inhaled cadmium to the general public than are most industrial emissions. Chronic cadmium exposure has caused hypertension in laboratory animals.

Sterekhova, N. P., A. A. Maratkanova, and I. K. Brodskaya. Experimental data on the combined activity of sulfur dioxide and dust at a copper smelting plant. *Gig. Tr. Prof. Zabol. 16*(3):52-54, 1972.

> Respiratory and digestive disorders are frequent occurrences in the copper smelting industry. The effects of sulfur dioxide and dust from industrial ash containing silicon dioxide, copper, iron, sulfur, zinc, calcium oxide, and lead were studied in rats. After four hours of daily exposure to sulfur dioxide in concentrations of 143.0 mg/m^3 for four months, a toxic effect manifested by dystrophic alterations in almost all systems and organs, particularly the liver, was noted. After exposure to the dust for the same period, sclerotic alterations developed in the bronchi and lungs. After exposure to combined activity, sclerotic processes in the lungs were more pronounced than in either of the former tests.

Ponteva, M., and L. Hansson. Lead and common gastrointestinal symptoms. *Duodecim 90*(5):335-341, 1974.

> The blood concentrations of lead, zinc, and copper in Finnish military recruits with gastrointestinal symptoms, and of controls who did not have such symptoms, were determined. The lead content in the blood of the group with the gastrointestinal symptoms was significantly different from the symptom-free group. No significant difference existed with respect to zinc and copper levels.

Babushkina, L. G., N. P. Sterekhova, and F. S. Kuzmina. Some indices of lipid metabolism in the blood of workers engaged in copper works [Russian]. *Ter. Arkh. 43*(9):100-104, 1971.

> Indices of lipid metabolism were determined in the blood of 94 copper smelter workers with toxicochemical hepatitis pneumosclerosis (in the presence of absence of hepatitis developing due to a chronic action of sulfur dioxide), and in the blood of silicosis patients and donors. All patients showed a significant increase of total lipids content compared to donors.

Colten, S. R. A review of the health hazards from copper exposure. *J. Occup. Med. 16*:621-624, 1975.

> Exposure to copper dusts and fumes can result in respiratory and/or dermatological effects. The prompt emetic effect of copper usually limits its toxicity when taken orally. However, when ingested in elemental or salt form, gastrointestinal and liver effects are observed (the

author suggests that high enough dust concentrations would probably cause similar effects). Some adverse reactions are diarrhea, nausea, vomiting, and abnormal liver functioning. Biopsy of hepatic tissues from one group of copper-salts-poisoned patients showed tissue mucous which was attributed to acute copper toxicity.

Hale, Ellen. Use of 'hazardous' herb widespread. *Tucson Daily Citizen*, Tuscon, Arizona, Wednesday, May 4, 1977.

> Use of a toxic herb mistaken for a medicinal tea plant may be very widespread in Arizona and could account for some of the state's high incidence of liver disease. Two Tuscon specialists, Dr. Alfred E. Stillman and pharmacologist Ryan J. Huxtable have found several close associations between adults who have drunk the herb in a tea and who now suffer from liver disease. Arizona has long had unexplainable levels of cirrhosis, about 10 percent higher than the national average, according to Huxtable.

COMMENTS:

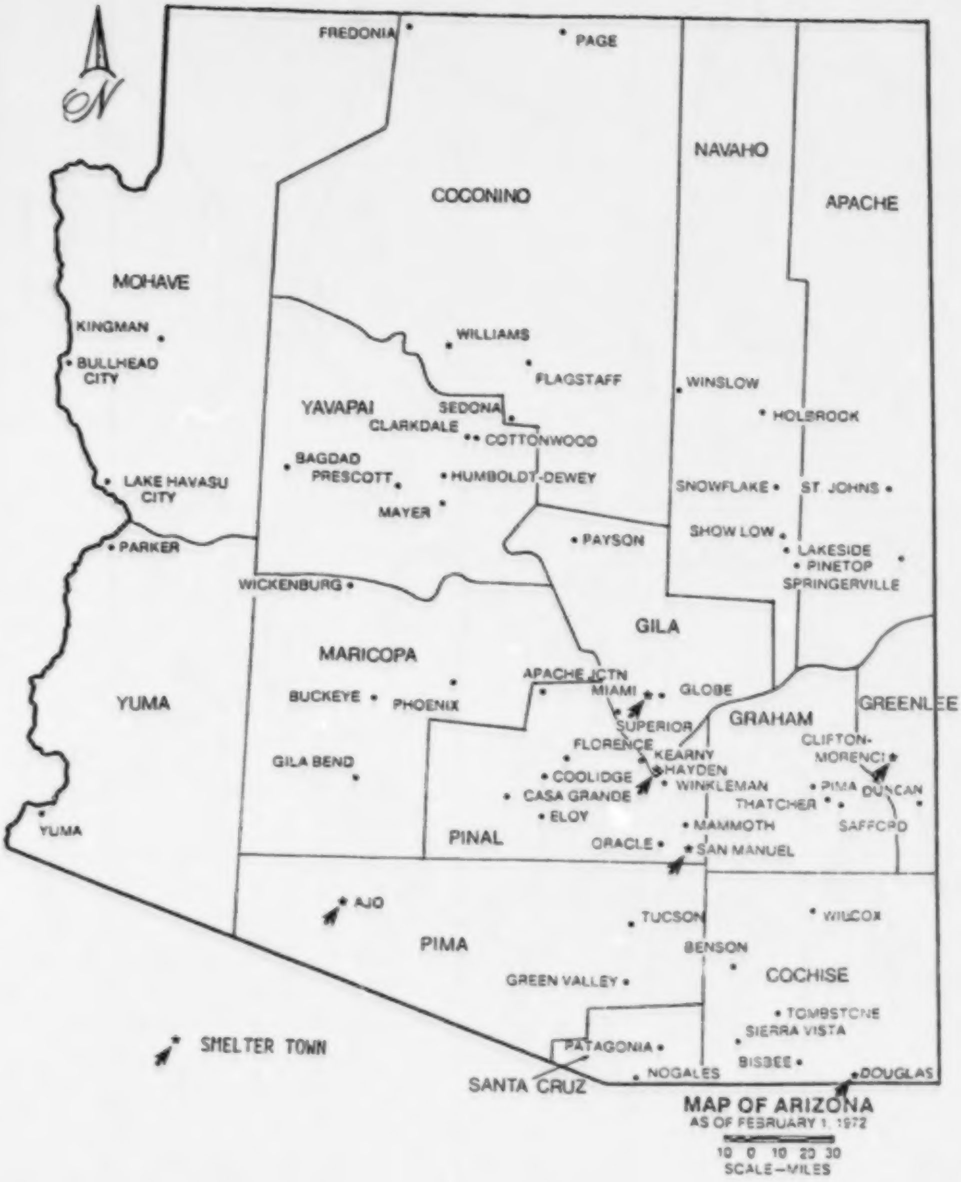
Copper smelter activity has been associated with adverse health effects, both among workers and in the community. Copper itself does not appear to be the toxic agent, but rather trace contaminants and gaseous wastes (in a few reported cases, ingestion of copper itself—in the form of salts—has been associated with liver abnormalities).

By-products and effluents of the industry which have appeared in the community and have been associated with disease are: arsenic (respiratory system cancer); cadmium (liver damage, cancer); and sulfur dioxide (liver disease). Furthermore, high levels of lead have been found in persons with gastrointestinal symptoms.

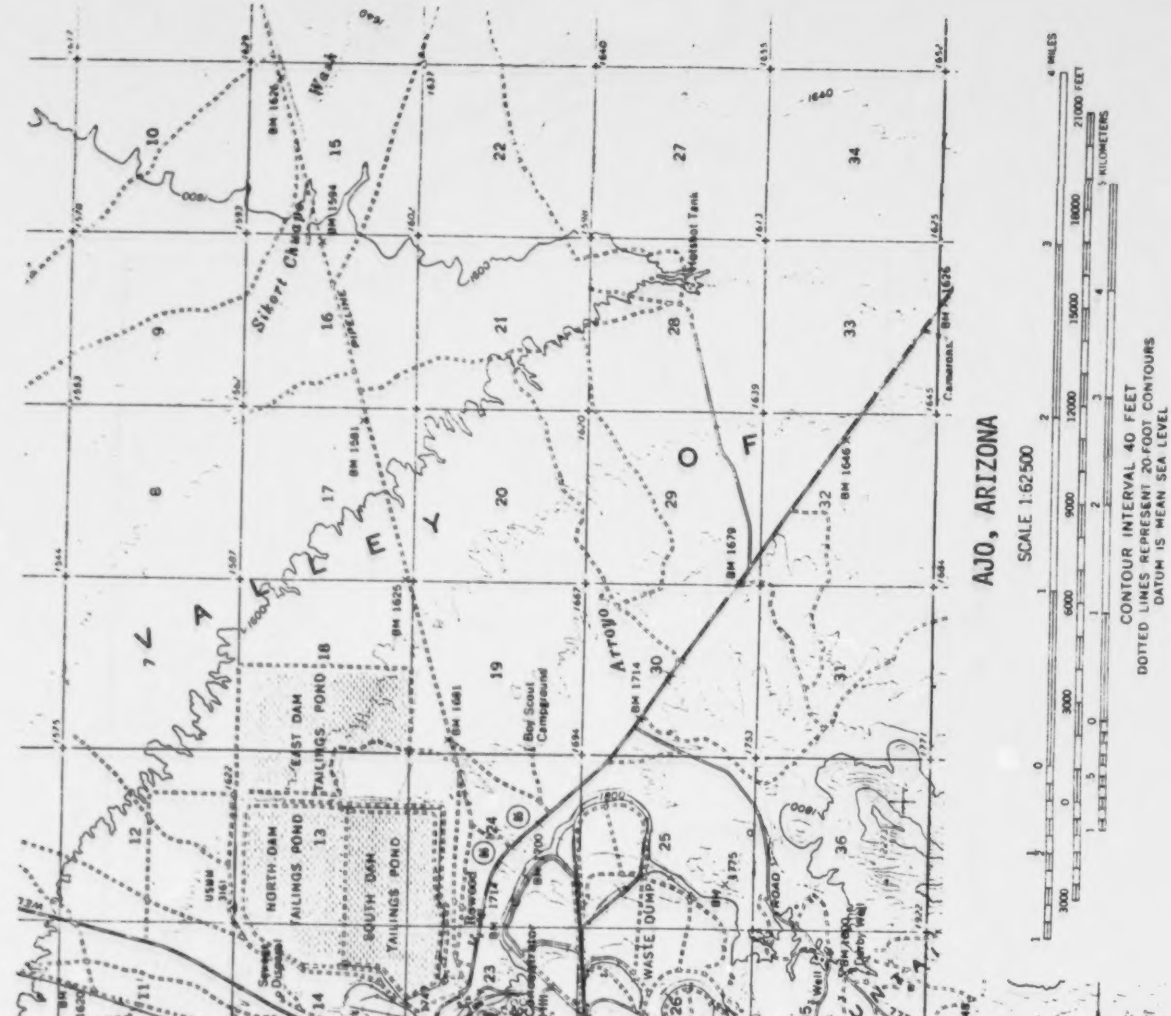
Appendix D

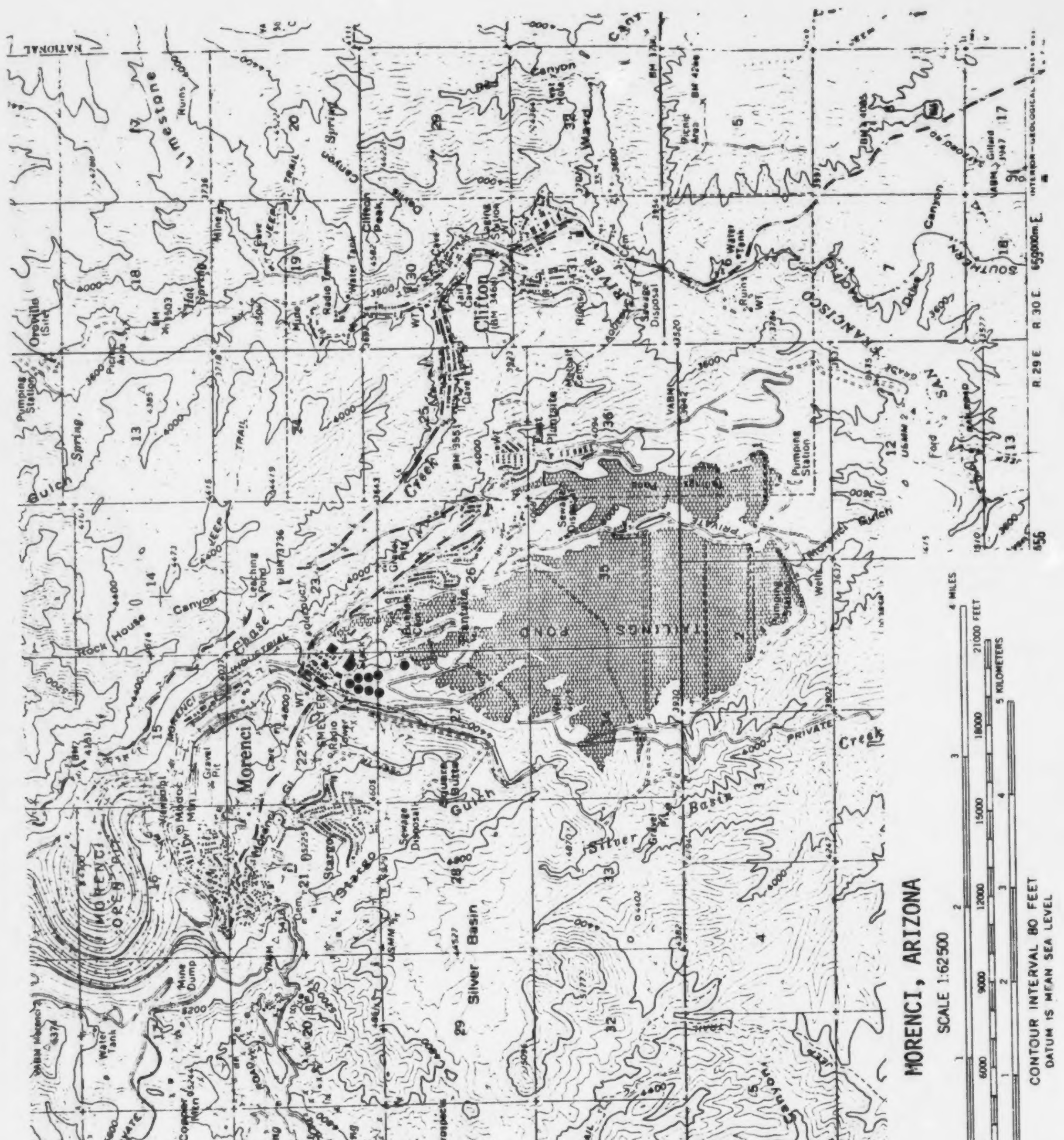
MAPS OF THE COPPER STUDY AREAS

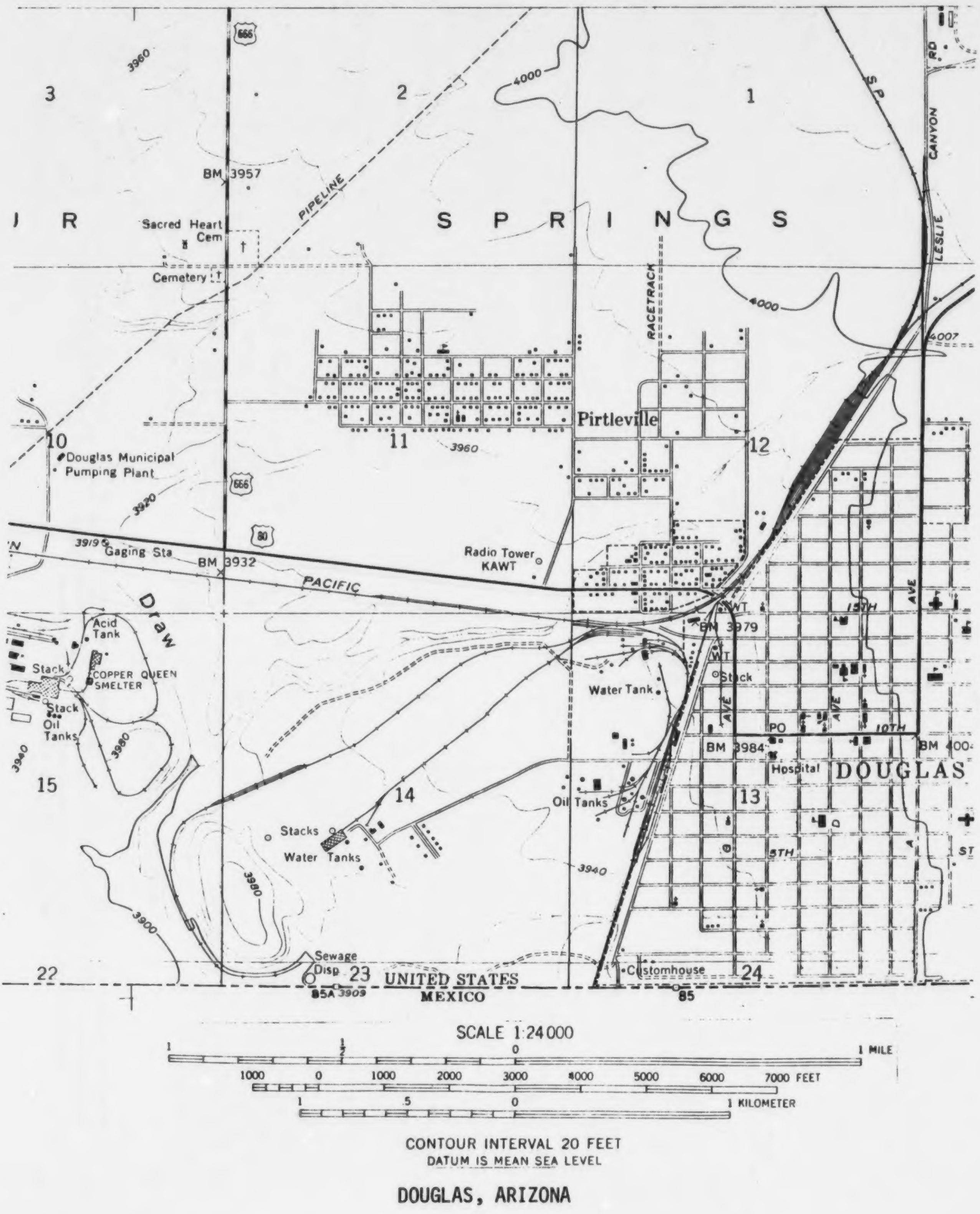
- Arizona
- Ajo, Arizona
- Morenci, Arizona
- Douglas, Arizona
- San Manuel, Arizona
- Miami-Inspiration, Arizona
- Hayden, Arizona
- Montana
- Anaconda, Montana
- Magna-Salt Lake Area, Utah
- Magna, Utah



MAP OF ARIZONA
AS OF FEBRUARY 1, 1972

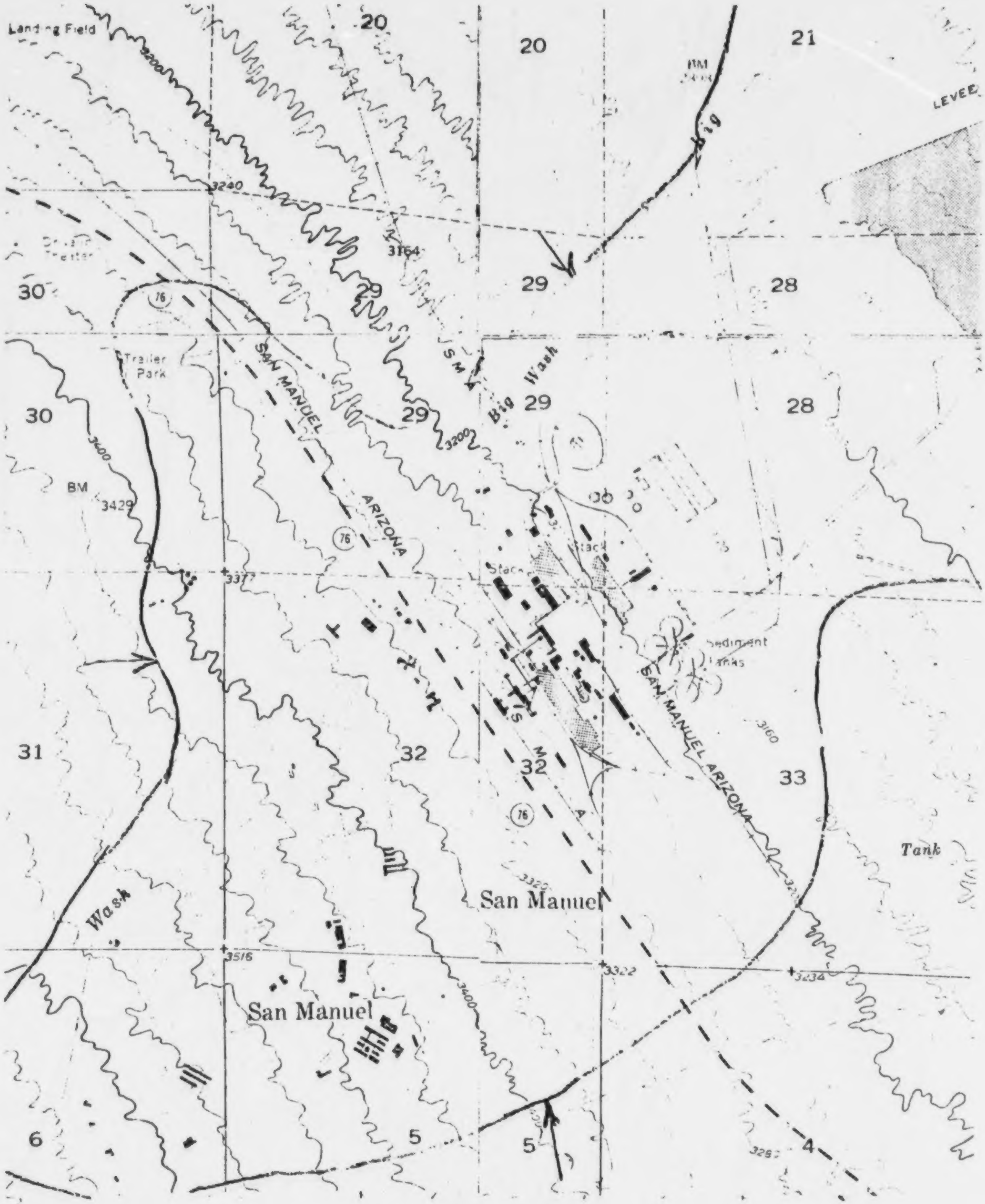

AJO, ARIZONA
SCALE 1:62500
CONTOUR INTERVAL 40 FEET
DOTTED LINES REPRESENT 20-FOOT CONTOURS
DATUM IS MEAN SEA LEVEL
MILES
FEET
KILOMETERS
NORTH DAM TAILINGS POND
SOUTH DAM TAILINGS POND
EAST DAM TAILINGS POND
WASTE DUMP
NEW CORNELIA
AJO
Gibson
Sikort Chuapo Wash
Arroyo
PIPELINE
Boy Scout Campground
Rowood
Concentrator
Hotshot Tank
Camelback Mtn
Gibson Arroyo
Pinnacle Peak
Cardigan Peak
North Ajo Peak
Ajo Peak
Darby Well
Darby Arroyo
Locomotive Rock
Valentine Well
Dunns Well
Well
ROAD
Cameron
BATES
Sewage Disposal
Drive-in Theater


MORENCI, ARIZONA
SCALE 1:62500
CONTOUR INTERVAL 80 FEET
DATUM IS MEAN SEA LEVEL
4 MILES
21000 FEET
5 KILOMETERS
Morenci
Clifton
MORENCI OPEN PIT
TAILINGS POND
Silver Basin
SMELTER
Chase Creek
SAN FRANCISCO RIVER
Limestone
Rock House Canyon
Leaching Pond
Pumping Station
Sewage Disposal
Mine Dump
Water Tank
Gold Gulch
Eagle Creek
Ward Canyon
Oroville (Site)
R. 29 E.
R. 30 E.
NATIONAL



CONTOUR INTERVAL 20 FEET
DATUM IS MEAN SEA LEVEL

DOUGLAS, ARIZONA



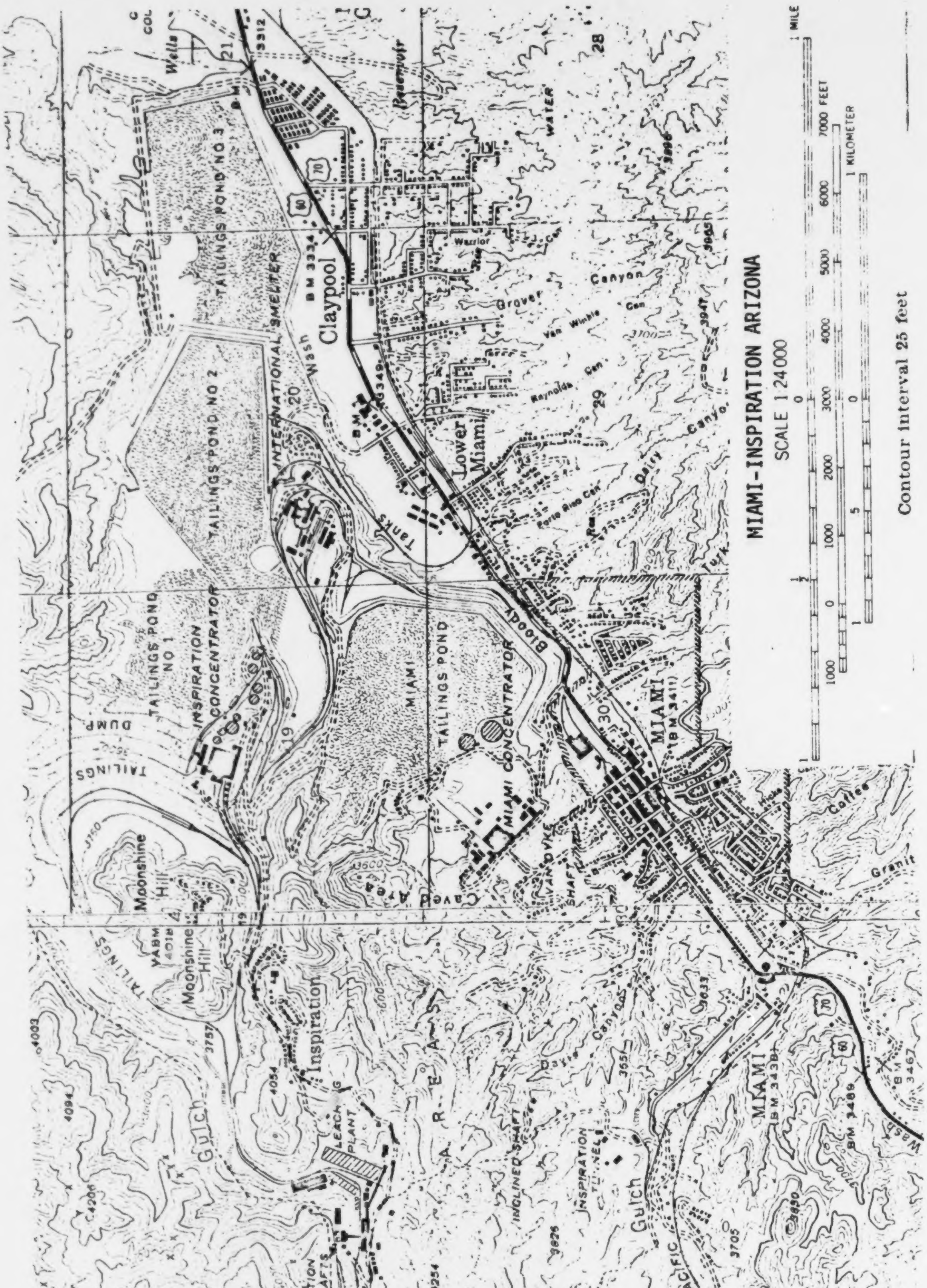
SAN MANUEL, ARIZONA

SCALE 1:24 000

1 ½ 0 1 MILE

1000 0 1000 2000 3000 4000 5000 6000 7000 FEET

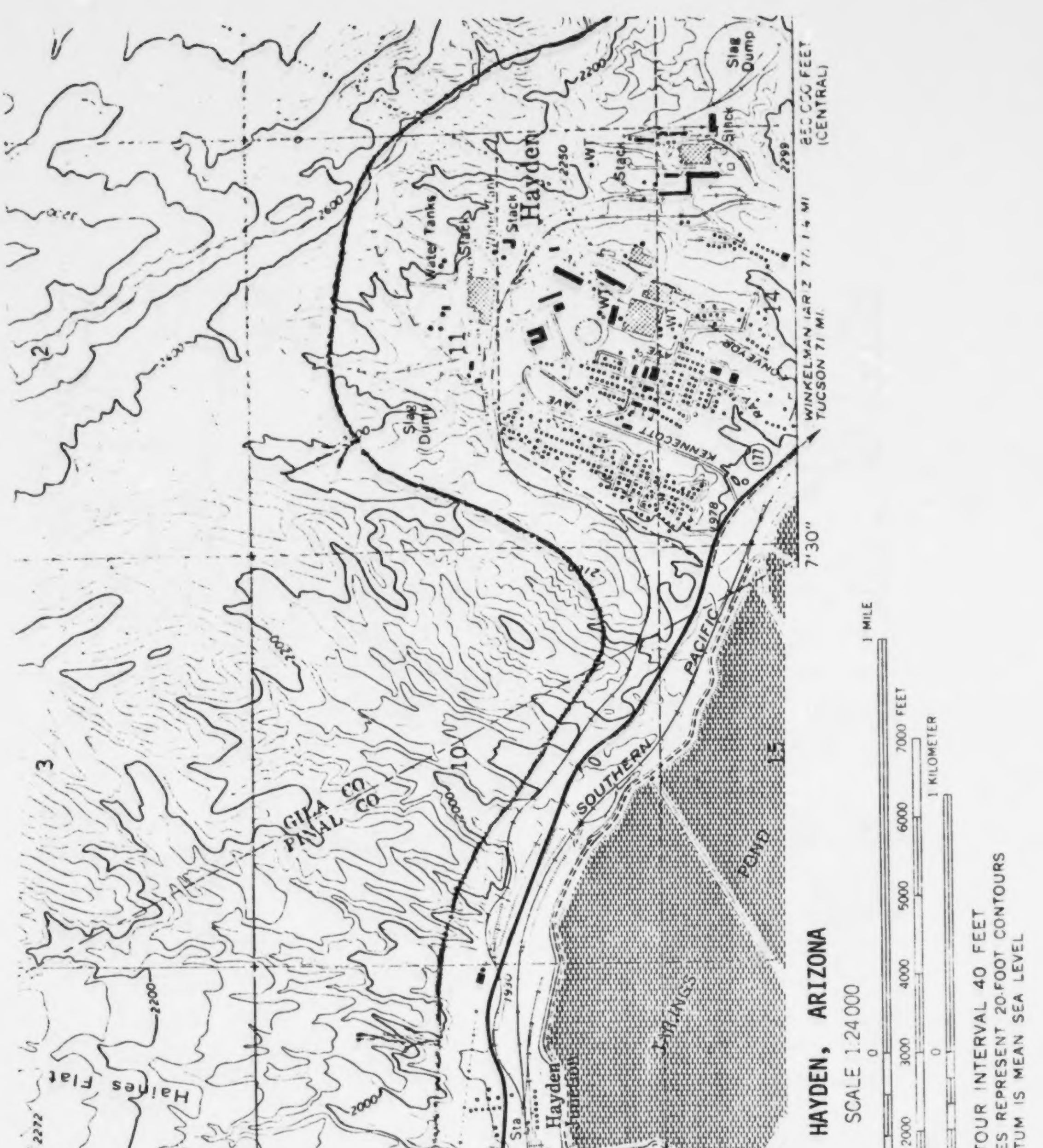
1 .5 0 1 KILOMETER

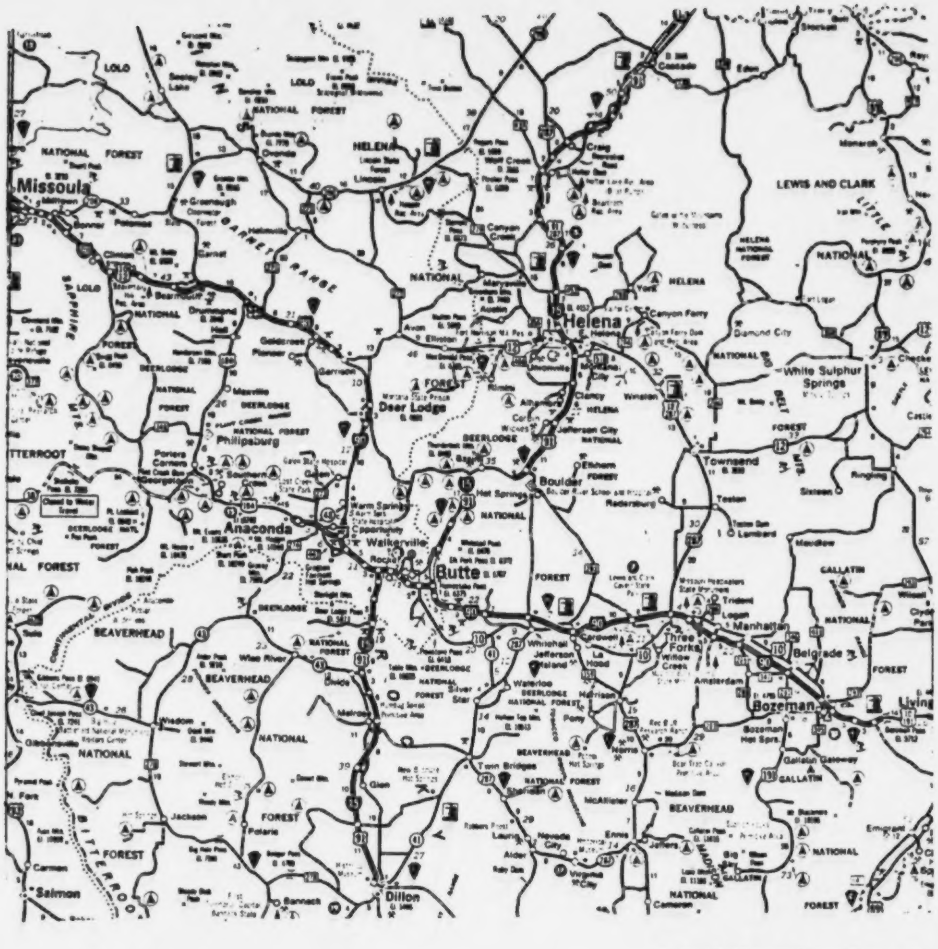


MIAMI-INSPIRATION ARIZONA

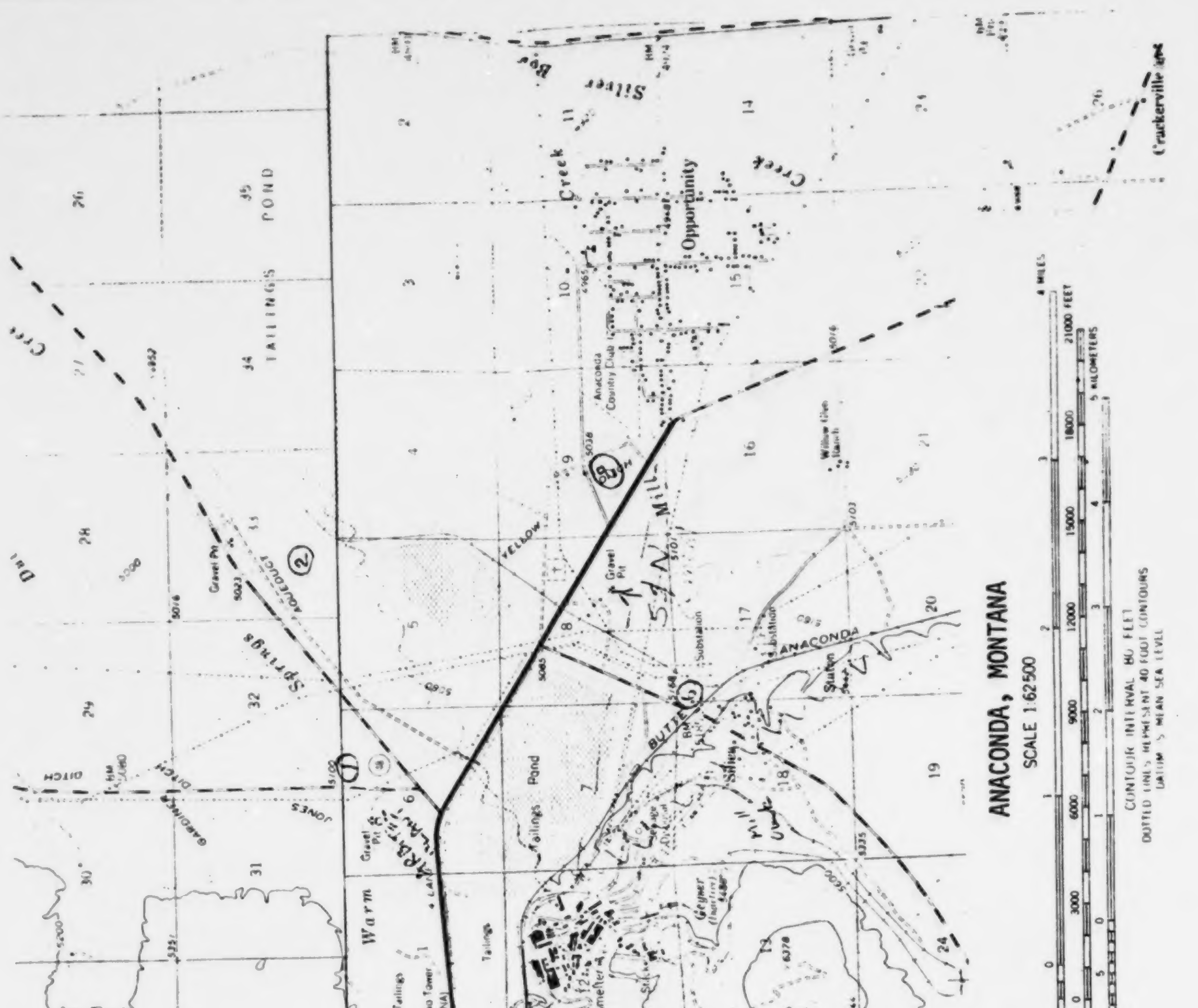
SCALE 1:24000

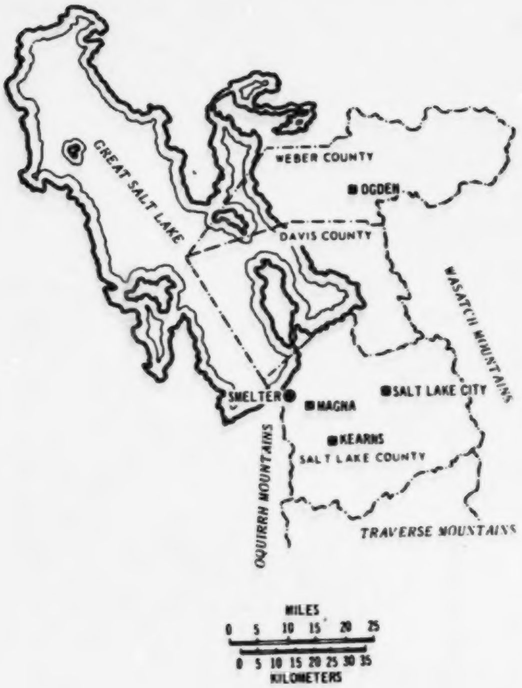
Contour interval 25 feet


HAYDEN, ARIZONA
SCALE 1:24 000
1 MILE
7000 FEET
1 KILOMETER
CONTOUR INTERVAL 40 FEET
DOTTED LINES REPRESENT 20-FOOT CONTOURS
DATUM IS MEAN SEA LEVEL
Haines Flat
GILA CO
PINAL CO
RIVER
Hayden Junction
Sta
Water Tanks
SOUTHERN PACIFIC
TAILINGS POND
Slag Dump
Stack
Hayden
WT
KENNECOTT AVE
RAY CONVEYOR
WINKELMAN (AZ 77) 1.4 MI
TUCSON 71 MI
850 000 FEET (CENTRAL)
7'30"
177
2
3
4
9
10
11
14
15
16
2200
2000
2272
1890
1930
1978
2250
2299

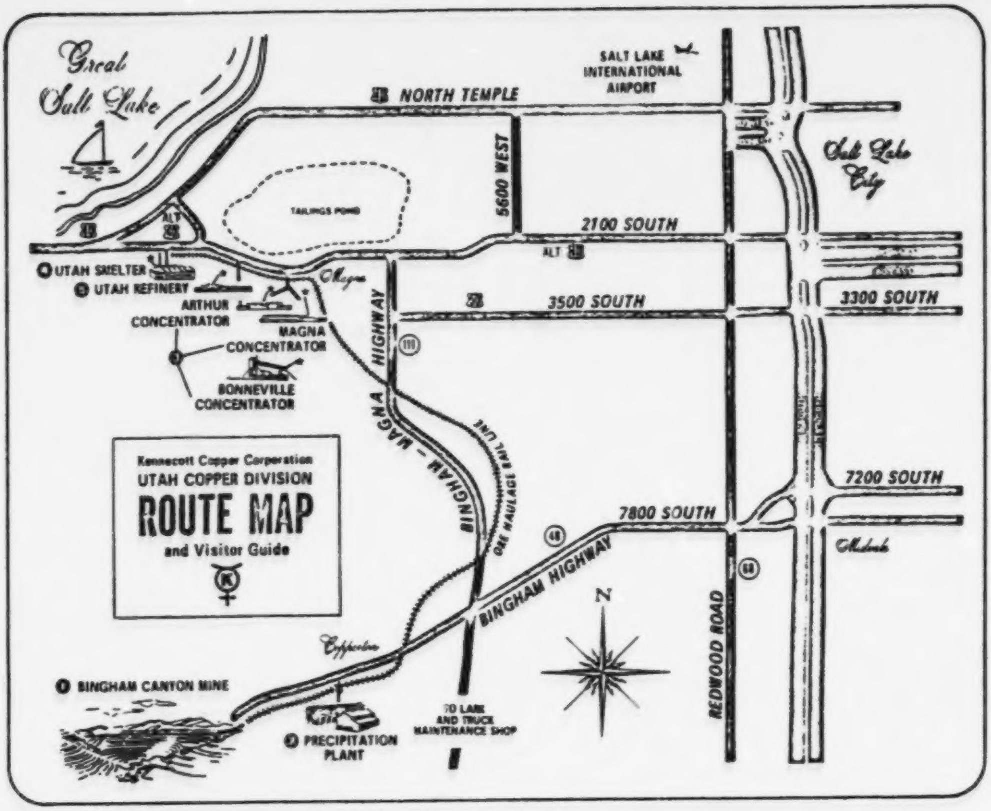

Missoula
LOLO
NATIONAL FOREST
HELENA
GARNET RANGE
Helena
LEWIS AND CLARK
White Sulphur Springs
Deer Lodge
Philipsburg
DEERLODGE
Anaconda
Walkerville
Butte
Boulder
Townsend
Three Forks
Manhattan
Belgrade
Bozeman
BEAVERHEAD
GALLATIN
Wisdom
Jackson
Dillon
Twin Bridges
Ennis
Virginia City
Salmon
0 5 10 20 30 40
One inch represents approximately 22 miles

MONTANA


ANACONDA, MONTANA
SCALE 1:62500
CONTOUR INTERVAL 80 FEET
DOTTED LINES REPRESENT 40 FOOT CONTOURS
DATUM IS MEAN SEA LEVEL
ANACONDA
Opportunity
Anaconda Smelter
Anaconda Country Club
Warm Springs
Tailings Pond
Tailings
TAILINGS POND
Silver Bow Creek
Mill Creek
Clear Creek
Sheep Gulch
Willow Glen Ranch
Crackerville
Ridge
Radio Tower
Gravel Pit
Substation
Jones Ditch
Yellow Ditch
Aqueduct
1
2
3
4
5
10
MILES
FEET
KILOMETERS



MAGNA-SALT LAKE AREA



MAGNA, UTAH

The copper production cycle starts at the Bingham Canyon Mine ①, where 108,500 tons of ore and over 300,000 tons of overburden, or waste material, are removed daily. Because overburden contains trace amounts of copper, it is treated with a solution which s subsequently processed at the precipitation plant ②. Ore hauled by train from the mine is processed through three concentrator plants ③ where it is crushed, ground and concentrated to nearly 30 percent copper. The smelter ④ takes the concentrate and produces blister copper, 98.6 percent pure; then copper anodes 99.5 percent pure. At the electrolytic refinery ⑤ the copper is further purified to 99.96 percent and cast into various shapes for Kennecott customers.

Appendix E

HISTORY OF AREA DEMOGRAPHY AND INDUSTRIAL ACTIVITY IN GILA AND PINAL COUNTIES OF ARIZONA

Appendix E

HISTORY OF AREA DEMOGRAPHY AND INDUSTRIAL ACTIVITY IN GILA AND PINAL COUNTIES OF ARIZONA*

GENERAL

Gila and Pinal Counties of Arizona constitute one of the study areas for the investigation of the relationship between the copper smelting industry and community health effects. There are presently four copper smelters in the area.

Copper mining and the related production of silver, gold, zinc, and molybdenum have been the basic forms of mineral resource utilization and the major impetus for the growth and development of the communities within the study area. Within Gila and Pinal Counties in general, growth patterns have been greatly influenced and determined not only by the mining activities but also by the fact that a significant amount of land is unavailable for residential development. A combination of vast expanses of government-controlled land, the extensive Indian reservations, and the large private land holdings controlled by the mining and ranching interests have helped channel area growth. Today, as in years past, populations within the study area are in close proximity to mining and smelter activities and are clustered into discrete towns separated by stretches of uninhabited desert.

With the exception of Winkelman, all of the study area communities were founded originally as camps associated with either silver or copper mining activities. Today the strong influence of copper mining is evident throughout the study area; i.e., the distribution and patterns of employment, the characteristics of the labor force, the types of commercial service establishments, and the kinds of trade goods offered for sale are reflective of an area with one major economic base. All of the urban communities are oriented towards servicing this mining base either directly or indirectly. As a result of this total dependence upon mining, the size of the various communities fluctuates in response to the economic status of the local industry. This association was particularly evident in the early 1930's during the depression, when several mines were shut down and many miners were forced to leave their homes or find jobs in the few area mines that remained open. During that period most of the study area communities decreased in population, as indicated in Table 1 which presents population statistics for the study area for the past fifty years.

Extensive amounts of historical information concerning the communities are not readily available. The primary sources used to obtain community and industrial historical data were as follows:

- Chamber of Commerce for each community
- Arizona Office of Economic Planning and Development
- U.S. Department of the Interior, Bureau of Mines
- Library of Congress
- U.S. Department of the Interior, Geological Survey
- U.S. Department of Housing and Urban Development

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*This appendix was prepared by Susan A. Perlin.

Table 1. *POPULATION CHARACTERISTICS OF STUDY AREA*

TOWN	POPULATION							CHANGE IN POPULATION (Δ%)				
	1976[a]	1970	1960	1950	1940	1930	1920	1960-1970	1950-1960	1940-1950	1930-1940	1920-1930
SMELTER TOWNS												
Inspiration	N.D.	200	400[b]	500[c]	600[d]	500[e]	18[e]		-20.0	-16.7	+20.0	+2677.8
Hayden	1,300-1,500	1,283	1,760	1,494	3,000	3,500[e]	4,000[e]	-27.1	+17.8	-50.2	-14.3	-12.5
San Manuel	f	4,332	4,524	(-)	(-)	(-)	(-)	-4.2		--	--	--
Superior	5,400-5,700	4,975	4,875	4,500[c]	4,000[d]	5,500[e]	3,000[e]	+2.1	+8.3	+12.5	-27.3	+83.3
Miami	3,650-3,900	3,394	3,350	4,329	4,722	7,693	6,689	+1.3	-22.6	-8.3	-38.6	+15.0
TOWNS WITHIN 10 MILES OF SMELTER												
Claypool	N.D.	2,245	2,505	2,000[c]	1,800[d]	2,000[e]	N.D.	-10.4	+25.3	+11.1	-10.0	--
Globe	6,450-6,600	7,333	6,217	6,419	6,141	7,157	7,704	+18.0	-3.1	+4.5	-14.2	-7.1
Central Heights	N.D.	2,289	2,486	N.D.	N.D.	N.D.	N.D.	-7.9				
Kearny	2,800-3,000	2,829	902	(-)	(-)	(-)	(-)	+213.6	--	--	--	--
Winkelman	1,100-1,300	974	1,123	548	524	729	573	-13.3	+104.9	+4.6	-28.1	+27.2
Oracle	f	2,237	500[b]	500[c]	500[d]	200[e]	150[e]	+347.4	0.0	0.0	+150.0	+33.3
Mammoth	f	1,953	1,913	500[c]	650[d]	150[e]	450[e]	+2.1	+282.6	-23.1	+333.3	-66.7

[a] Population estimates from the Arizona Office of Economic Planning and Development, *Mid-1976 Population Estimates for Non-Metropolitan Arizona Communities* (no date).

[b] *Arizona, The Grand Canyon State; A State Guide*, Revised by Joseph Miller, Hastings House (American Guide Series), New York, 1966.

[c] *Arizona Business and Professional Directory, Vol. 17, 1951-53*, Baldwin and Mullin-Kille Company, Phoenix, 1951.

[d] *Business and Professional Directory of Arizona, 1941-42*, Arizona Directory Company, 1941.

[e] *Arizona State Business Directory*, 1920 and 1930 editions, Gazetteer Publishing and Printing Company, Denver, 1920, 1930.

[f] 1976 population estimate fo. Mammoth/Oracle/San Manuel: 9,700-9,900 people.

(-) = town not in existence.

N.D. = no data found.

- U.S. Securities and Exchange Commission
- Arizona State University (University Library), Tempe, Arizona
- State of Arizona, Department of Library, Archives and Public Records

Table 2 highlights the chronology of important events concerning the founding of communities and the operation of copper industries within the study area. As indicated in this table, all communities (with the exception of Kearny and San Manuel) have had a very long history of exposure to emissions and effluents associated with the various phases of copper mining and processing.

Table 2. CHRONOLOGY OF EVENTS

1867	-- Mammoth established as mining camp for area gold mines.
1870's	-- Winkelman founded as ranchers move into area. Economy originally based on serving the area agricultural sector.
1876	-- City of Globe established to serve the area silver mines.
1880	-- Oracle established as a supply center for several mines in nearby Santa Catalina Mountains.
ca. 1880	-- Superior founded as mining camp near site of Silver Queen silver mine.
1881	-- Old Dominion Copper & Smelting Company starts operations at Globe and builds a "small smelter" (2). -- Small copper furnace starts operations at present site of Miami (10).
1883	-- Some copper mining started at Ray deposits, near present site of Hayden (10).
1898	-- Old Dominion Company builds new smelter at Globe, and for the next 12 years the Old Dominion mine remains one of the world's "greatest copper mines" (2).
1900	-- Smelter built at Kelvin to treat copper ores from Ray and Miami mines. Processed over 16,000 tons of ore and later shut down (11).
1907	-- Extensive development of Ray copper deposits (10). -- Townsite of Miami established as a copper camp (2).
1909	-- Hayden founded as milling and smelter camp for area copper mines. -- Inspiration Mine Company starts constructing huge reduction plant at Miami.
1911	-- Copper production starts at the Miami Copper Company deposits. -- ASARCO builds copper smelter at Hayden to treat Ray copper concentrates and custom ores. -- Ray mines copper production starts on large scale; concentrating mill completed with 8,000 ton per day capacity. -- Copper production starts at Magma Copper Company Superior location (10).
1912	-- Miami Copper Co. completes concentrator, bringing capacity to 3,000 tons of ore per day (11) -- Discovery of rich copper ore at Superior in the mines which for several previous years had been producing silver (11).
1915	-- Flotation process introduced at Inspiration and becomes the first large-scale copper flotation plant in U.S.A., with capacity of 14,000 tons of ore per day (10). -- International Smelting Company erects new smelter at Miami to handle the increased load of copper concentrates resulting from completion of two new mills at Miami and Inspiration (11).

(continued)

Table 2 (continued). CHRONOLOGY OF EVENTS

1923	-- Miami Copper Co. increases mill capacity from 3,000 to 12,000 tons of ore per day and improves block caving methods to increase ore production (11).
1924	-- Smelter facility completed by Magma Copper Company at Superior (10). -- Development started of low-grade ore at Miami.
1926	-- Large-scale leaching and precipitation operations started at Inspiration—built 9,000 ton leaching plant (11).
1927	-- Miami Copper Co. increases mill capacity from 12,000 to 18,000 tons of ore per day (11).
ca. 1930	-- Depression of early 1930's has terrific impact on all copper mines in Arizona, with many forced to close for various lengths of time (11). -- Old Dominion mine and smelter at Globe shuts down (2).
1939	-- Advent of World War II causes copper prices to rise and increases production in Arizona.
1943	-- Open-pit copper production starts at Castle Dome mine, owned by Miami Copper Co. and located six miles west of Miami. Built concentrator with 12,000-ton per day capacity. (Venture backed by U.S. Government for war needs.) (11)
1948	-- Open-pit development is completed at Inspiration and Ray mines (10). -- Underground exploration starts at San Manuel (10).
1950	-- Open-pit production starts at Ray mines (10). Prior to this, production was all underground, with 79 million tons of ore mined from 1911-1954 (11).
1951	-- Kennecott installs pit-crushing plant at Ray and expands its milling facilities at Hayden in order to maintain ore production and treatment at 15,000 tons per day. ASARCO's Hayden plant smelts the concentrate.
1953	-- Castle Dome ore reserves exhausted; mine closed after producing 257,000 tons of copper plus gold and silver (11). -- Development started of Miami's low-grade ore (10). -- Asbestos purchase depot opened by U.S. Government at Globe (10). -- Copper Cities deposit (three miles north of Miami and mined by Copper Cities Mining Co., a subsidiary of Miami Copper Co.) comes into production (11).
ca. 1954	-- Community of San Manuel established as a new mining camp associated with development of the San Manuel copper ore deposits.
1954	-- Miami starts production from low-grade copper ore body (10). -- Concentrator moved from Castle Dome to Copper Cities mine site. -- Ray mine stops underground mining and becomes completely open-pit operation. To handle increased ore production, the concentrator capacity is increased to 15,000 tons per day (11).
1956	-- San Manuel starts underground mining, flotation concentration and smelting of concentrates (19). Flotation concentrator has 30,000-ton capacity (11)
1960	-- Inspiration Consolidated Copper Co. purchases International Smelting and Refining Co. smelter located at Miami. At time of purchase, smelter already treating Inspiration concentrates, cement copper and material from toll customers (18).
ca. 1960	-- Community of Kearny built for the Ray Mines Division to house workers at neaby open-pit mines and reduction plant. -- Capacity of Ray mine's concentrator increased to 22,500 tons per day (11).
1962	-- Copper Cities mine, since opening in 1953, has produced over 27 million tons of ore at a daily rate of 12,000 tons (11). -- Inspiration's Christmas mine to start production in mid-1962.
ca. 1963	-- Miami Copper Co. has exhausted mineable grade ore in their underground Miami mines. To recover remaining copper, the entire underground network was flooded with a leaching solution (note: roughly 10 years later, in 1973, copper was still being recovered by this leaching process) (20).

(continued)

Table 2 (continued). CHRONOLOGY OF EVENTS

1967	-- Bluebird mine, between the Inspiration and Castle Domes mines at Miami, produced 4,100 tons of cement copper. Mine owned by Ranchers Exploration and Development Co. (11).
1969	-- Magma Copper Co. starts program to increase ore production at San Manuel underground mine from 40,000 to over 60,000 tons per day by 1972. Plans started for electrolytic refinery at San Manuel with capacity to extract 200,000 tons of refined copper per year starting 1972 (21).
	-- Magma Copper Co. starts program to increase ore production at Superior from 1,500 to 2,500 tons per day by 1974 by expanding mine and mill facilities. Plans call for ultimate closing of Superior smelter and shipment of concentrates to San Manuel (21).
	-- Kennecott Copper Corp. Ray Mines Division completes electrowinning plant to produce refined cathode copper from solutions obtained at its newly-built silicate ore, vat leaching plant at Ray. Electrowinning plant designed to treat 10,000 tons of ore daily also includes contact acid plant with designed capacity of 750 tons per day (21).
	-- Inspiration Consolidated Copper Co. starts production of copper rod at its new mill in Miami.
1971	-- ASARCO installs sulfuric acid plant at Hayden smelter to reduce SO_2 emissions.
1973	-- Inspiration Consolidated Copper Co. completes air pollution control project (to be fully operational in 1974), consisting of building a new smelter with the world's largest electric smelting furnace, new siphon-type converters, a waste gas cleaning plant and a sulfuric acid plant. Copper production will eventually reach annual rate of 10 million pounds (20).
	-- Kennecott completes installation at their Hayden smelter of an SO_2 air quality control system having an expanded 900 ton per day sulfuric acid plant, improved converter hoods and gas collector, and a complex computer control system (14). When the system is fully operational in 1974, it is expected to bring smelter into compliance with all ambient air quality standards (22).
	-- Magma's San Manuel mine was largest producer in Arizona.
	-- At San Manuel smelter, installed new water-cooled converter hoods as part of air quality control program. Made plans for neutralization plant to dispose of sulfuric acid which will be a by-product of the new sulfuric acid plant, designed to convert SO_2 gas from the smelter into acid. Acid plant projected to be completed in summer of 1974, with daily production of 2,000 tons of sulfuric acid at full production. This smelter chosen by the Smelter Control Research Association as a test site for proposed experimental plant control SO_2 generated by reverberatory furnaces (20).
	-- Magma's Superior mine reached daily production of 3,000 tons from deeper high-grade ore body of 4.2% copper (20).
	-- ASARCO smelter at Hayden modified by addition of anode casting plant to eliminate casting of blister copper cakes that previously were remelted and cast into anodes at the refinery. Also built 1,000-foot stack to discharge weak SO_2 gases that cannot be handled by the acid plant (14). Anticipated that when stack is fully operational in 1974, ambient air quality will improve (22). ASARCO also installing "closed loop" SO_2 monitoring system at smelter that monitors ambient air quality and indicates when SO_2 emissions exceed allowable limits so that smelting can be curtailed to adequately control emissions (22). This system is jointly owned by ASARCO and Kennecott (23).
	-- Kennecott initiates plans to expand Ray mines silicate leach plant from 10,000 to 14,000 tons of ore per day, with 14 months scheduled for completion (22).
1974	-- Cities Service's Pinto Valley open-pit mine (eight miles west of Miami) starts production with expected daily capacity of 40,000 tons of ore by year's end. Capacity at start-up is 20,000 tons of ore per day. The facility has a mill and sends concentrates to the Inspiration smelter (20).
	-- Kennecott Copper Corp. Ray Mines Division received the first unconditional smelter-operation permit from the Air Pollution Control Division as a result of removing over 90% of smelter particulates and SO_2 emissions.

HISTORY OF COPPER COMMUNITIES

Globe

Globe is located in a steep canyon, just south of the rugged Salt River Canyon, at an elevation of 3,500 feet above sea level. A number of hills and mountain ranges with elevations from 4,000 to 8,000 feet surround the Globe-Miami area.

The community, today the County Seat for Gila County, was once a typical example of the boom and bust company mining towns. Originally settled in 1876 as the result of a silver strike, the community really came into its greatest wealth and prosperity starting in the late 1880's after the silver had been exhausted and the rich copper deposits were discovered. The Old Dominion Copper Company was established in the late 1880's with a "small smelter," but this was soon replaced by a new, larger unit in 1898. For the next 12 years, or until around 1923, the Old Dominion was "one of the greatest copper mines in the world." Other mines developed in the area and there was extensive cattle grazing in Gila County, with much of the income from these two businesses spent or invested in Globe. The town flourished and called itself the "Capital City of the County with a Copper Bottom" (2).

The decline of Globe started in the early 1900's as a result of miners' strikes, drought, overgrazing of the range, and the establishment of new copper mines and a new camp in nearby Miami. The 1931 Depression closed many area mines including the Old Dominion operations, and for two years most of Globe's men worked for the WPA (Work Projects Administration). The Old Dominion never reopened because of outdated equipment, very low grade ore, and flooding of the mine. When operations started up again in Miami, many of Globe's miners found work there. Globe has remained a quiet town ever since.

Today Globe is a trade and service center for the surrounding areas of southern and central Gila County.

A description of the community written in the early 1950's indicated that the mining and smelting activities had had a long history of impact on the area. At the northern end of town "black slag dumps and weathered tailings dumps (gigantic accumulations of copper ore residue)" were the remains of a once great copper mine (2). The waste material pushed out on the flat plateaus and into the gulch of Pinal Creek and formed man-made hills along the road.

Apparently by the 1950's the town's appearance had not changed significantly from the original old-fashioned western mining town. Now, as in the past, the town attracts many Apache Indians from nearby San Carlos Reservation. Even in the early days when the community was just a mining camp, the population was a mixture of many nationalities including English, Mexicans, Italians, and Slavs (2).

Presently, Globe, along with many of the other communities in the study area, faces the problem of lack of adequate housing available for residents. A 1970 survey found that slightly over half of the town's 2,899 housing units were deteriorating or dilapidated, and that approximately 300 families were living in substandard housing and were unable to afford anything better (6).

Miami-Inspiration/Central Heights

Miami is situated near Globe, at an elevation of 3,500 feet above sea level, in the foothills of the Pinal Mountains. As noted in the description of Globe, Miami lies between the walls of a fairly steep canyon.

In 1907 the camp of Miami was established approximately seven miles west of Globe beside low-grade copper deposits that were much more extensive than those of Globe's Old Dominion mine. Originally the huge deposit was mined by two companies—i.e., the eastern part by the Miami Copper Company and the western part by the Inspiration Consolidated Copper (ICC) Company. In 1912 the ICC Co. began construction of its huge reduction plants, and from 1915 to 1920 the Miami district was producing to the limit of its capacity. The community spread out into the suburbs of Lower Miami and neighboring Claypool and supported a population that was growing larger than that of its rival, Globe.

Then, as now, the town's business section was on the valley floor, and most residences were located in the hills and canyons surrounding the floor. For a long time the area supported a mixture of flimsy homes, typical of mining camps, and some better residences. On the hills to the north of town, company villages and the huge copper plants have been existing for over 30 years. A very graphic description of the area, written in the early 1950's, vividly portrays the industry's effect on the town:

> "The smelter's coal-black slag dumps and cement-colored tailings from the concentrators stretch for miles over the heights and down into the flat alongside the town. At night when the smelter is working, the mounds of slag glow red with streams of molten rock dumped from the furnaces." (2)

In the 1920's Miami was a rowdy community with over 40 gambling halls and brothels. Today it is a far more quiet place with roughly half the population size. A 1970 survey found 54 percent of the town's 2,775 housing units deteriorated or dilapidated, with some in such bad condition that clearance and redevelopment were suggested as the most feasible means of providing safe, decent housing. The survey also found the majority of commercial buildings in the business district were vacant, deteriorating and/or obsolete (6). For many years now there has been little construction and residential growth in Miami.

Up to 1971 the most recent development in the Miami area had taken place in the Central Heights and Corbe Valley districts. Little Acres, Inspiration, and Miami Gardens were predominantly company housing projects (built and maintained by Inspiration Consolidated Copper Co.) and offered homes that were in better than average condition (6).

In the 1950's Inspiration was noted as a well-kept company-owned village for the mining officials, supervisors, and skilled employees. Even then, the huge vats of the ICC leaching plant could be seen in the gulch below the community (2).

Prior to 1907 Miami was almost completely uninhabited by whites. As the town prospered, more than a dozen different nationalities were represented, with the Mexicans and Slavics forming the largest groups. After the postwar depression of 1921, the racial composition started shifting towards having a majority of Mexicans (2).

Mining activity across Arizona had been severely curtailed during the economic depressions of 1921 and 1931. Unlike some other mines in the area, those of Miami were able to resume full production in the post-depression periods. During the 1931 slump Miami Copper Co. curtailed production and ICC shut down almost completely, putting almost the entire town's population out of work.

During World War II, although hampered by strikes, Miami district mines produced copper to full capacity. To help the war effort, the Castle Dome mine, to the west of town, was brought into production and kept operating until the ores were exhausted in the 1950's.

Today Miami is the scene of extensive copper mining and processing activities (see Table 2). The ICC Co. has three open-pit mines, and smelter and rod fabrication plants. Ranchers Exploration and Development Corp. has open-pit and underground operations, plus leaching and concentrating facilities at their Bluebird mine. Cities Service Co., mining in the area since 1911, has a newly-developed open-pit facility, Pinto Valley, and a new underground mine, Miami East, in addition to its Miami mine. This firm also operates two concentrator and leaching facilities and an electrowinning plant.

The copper industry is the major employer, and other area activities such as cattle ranching, asbestos mining and processing, and sawmill operations offer a limited number of jobs to Miami residents.

Hayden and Winkelman

Hayden and Winkelman are located in the San Pedro Valley at the confluence of the San Pedro and Gila Rivers. The terrain is rolling desert with an elevation of approximately 2,000 feet above sea level.

Winkelman was originally founded in the 1870's as an agricultural community serving the agricultural businesses of the San Pedro and Gila River valleys, and also served as a shipping point for cattle raised in these valleys. Later, as the copper industry moved into the area, Winkelman became a trading center and residential area for employees of the mines and smelters (1).

Hayden was originally founded in 1909 as a milling and smelter camp for the area mines and remained a "company town" until 1956 when it was incorporated and became a self-governing community.

Productive mining in this area started in the early 1900's with the development of the Ray mines, now owned by Kennecott Copper Corp. Also in the early 1900's American Smelting and Refining Co. built a smelter in Hayden to treat concentrates from the Ray mines. In 1958 Kennecott also constructed a smelter at Hayden. Presently the mining and ore processing activities of these two companies dominate the economies of both communities.

For many years now, growth and development of these communities have been severely restricted since the mining companies own extensive landholdings in this area. With the exception of land presently occupied by homes and businesses, most of the remaining area is owned by the copper companies (6).

A 1970 survey of these two communities indicated that 59 percent of the housing stock (i.e., total of 741 units) were sound. The remainder were deteriorating or dilapidated, and many had excessive deterioration (6).

San Manuel and Mammoth

San Manuel and Mammoth are both located in the broad San Pedro River Valley at elevations ranging from 4,500 feet (i.e., Mammoth) to approximately 7,000 feet (i.e., San Manuel) above sea level.

San Manuel was built in 1954 by the San Manuel Copper Corp., a wholly owned subsidiary of the Magma Copper Company, as a mining camp for the employees of the newly established San Manuel mine, mill, smelter and refining complex. Originally the camp had approximately 7,000 residents and was described as a completely modern town with 2,000 homes plus parks, schools, churches, etc., that had sprung up in the desert overnight (2).

In the 1950's, when the mining was getting under way, the San Manuel mine was noted as one of the largest copper producers of the time. The company was then planning to spend over $100 million to create a producing capacity of over 70,000 tons of copper annually (2).

Mammoth, unlike San Manuel, was founded in the late 1800's as a gold mining camp and site of a stamp mill for gold taken from the area mines. The community is located near the St. Anthony deposit which was developed in the late 1800's as three separate properties—Collins Mine, Mammoth Mine, and Mohawk Mine. From its establishment in the 1800's to its exhaustion around 1953, this cluster of mines produced two million tons of a variety of ores including gold, silver, vanadium, molybdenum, lead, and zinc. In the early 1880's Mammoth was one of the "busiest and most lusty" of mining camps in the country. When the gold mill was moved out of town, and much later when the ores were depleted, Mammoth survived. The town has been sustained by residents in the valley, the stockmen, the agricultural sector, and by the fact that much money has been spent there by the San Manuel Copper Corp. and its employees for supplies, services, and recreation (1).

In the 1950's Mammoth was a quiet one-street town extending about one-half mile and having many homes and stores built of adobe, with many already falling into ruin (2).

When the St. Anthony deposit was active, the community of Tiger was one of the area's largest towns but its existence was totally dependent on the mines. When the ores were depleted in the 1950's, most of Tiger's residents were forced to move and most of them went to Mammoth (see Table 1). Today Tiger consists of a warehouse and storage area for some mining equipment (1).

Oracle

This community was founded in the 1880's as a supply center for several area mines. It is located in the San Pedro River Valley at an elevation of 4,500 feet.

Oracle provides homes for many of the area miners and for many years also has been a health resort and guest ranch community. Many rustic homes and guest ranches are in the hills surrounding Oracle.

Superior

Attention was first attracted to the Superior area in 1875 with the discovery of the rich Silver King mine. Subsequent opening of the nearby Silver Queen mine caused a great influx of people to the area. In 1910 the Magma Copper Company (today a subsidiary of Newmont Mining Corp.) formed to take over the Silver Queen properties, and 14 years later built a large smelter to process all of its ore plus hundreds of carloads of ore from smaller mines. The copper ore from the Magma mine was so rich that the mines and smelter were kept operational even during the slump of 1929-1933, when many mines throughout the country were shut down (2). The smelter at Superior was eventually shut down in 1971.

The community of Superior originally was established as a mining camp to house workers from Magma's underground mine. The commercial and service sectors primarily serve the mine employees in the immediate area.

Kearny

Kearny, located at an elevation of approximately 2,000 feet, is a completely planned community developed about 17 years ago for the Ray Mines Division of Kennecott to house workers at their nearby open-pit mine and reduction plant. Ranching, farming, and home building have a great influence on the economy of the community; however, the copper industry dominates the financial structure. The community is described as having a completely modern architectural design and no substandard housing (19).

The Ray mines and ASARCO's smelter at nearby Hayden provide most of the employment for Kearny residents. The commercial and service sectors provide a limited number of jobs.

HISTORY OF COPPER COMPANIES

Inspiration Consolidated Copper Corp.

This company owns and operates several mines including the Thornton, Live Oak, Red Hill, Black Copper, and Ox Hide mines around Inspiration and the Christmas open-pit and underground mines near Christmas. By-products of the mining operations include molybdenum, silver, gold, and selenium.

The Ox Hide mine is a heap-leaching operation. Ore is mined, placed on pads with impermeable bottoms, and leached with recirculated sulfuric acid solutions. Copper is recovered from solution by precipitation on scrap iron and then is trucked to Inspiration for smelting and refining.

The smelter located at Inspiration treats copper-bearing materials from the various Inspiration mines, plus custom and toll materials from outside customers. New copper-bearing materials treated in 1970 totaled 313,431 tons as compared with 316,591 tons in 1969.

At the Christmas mine, processing facilities include crushing and concentrating equipment, with the concentrates being sent to Inspiration for smelting and refining. At the remaining mines around Inspiration, facilities include crushing and leaching plants, concentrators, precipitation, electrowinning and electrorefining plants, smelter, and rod fabrication plants.

Around 1972 all the mines and plants were operating at a full daily capacity rate of roughly 42,000 tons of ore. At the copper content for that year, it was estimated that total company mine production would have been approximately 70,000 tons of copper per annum. At that time it was also estimated that the electrorefining plant and smelter were operating at capacity and producing approximately 45,000 tons of copper for return to toll customers. (Source: Company Annual Report, no date on our Xerox copy, but probably 1972.)

In 1951 the company owned 2,825 acres at Inspiration and had a 10,000-ton leaching plant and 15 unit concentrator with sliming department (15). Tables 3 and 4, extracted from references 15 and 16, give copper production figures for years 1948 through 1951 and years 1958 through 1961, respectively.

Table 3

SMELTER PLANT PRODUCTION, 1948-1951 (15,16)

(tons of copper)

	1948	1949	1950	1951
From Ores:				
• Electrolytic copper from cathode & starting sheets	31,317	26,264	29,446	30,297
• Blister copper in excess of starting sheet requirements	7,034	4,959	6,906	4,680
From Leaching-In-Place:				
• Blister copper	--	--	2,089	4,147

Table 4

SMELTER PLANT PRODUCTION, 1958-1961 (15,16)

(tons of copper)

	1958	1959	1960	1961
From Ores:				
• Electrolytic copper	37,511	41,659	35,324	36,616
• Blister copper	1,152	1,534	2,460	212
• Cement copper	--	--	75	397
From Leaching-In-Place:				
• Electrolytic copper	2,379	1,133	2,430	1,940
• Blister copper	778	2,685	112	--

In 1960 the company bought the International Smelting and Refining Co. smelter located at Miami. At the time of purchase the smelter already was treating Inspiration concentrates, cement copper, and material from toll customers. By the end of 1960 the company also had plans to expand their electrolytic refinery at Inspiration. This enlargement plus the smelter purchase was to enable the company to ship the entire output of its mines and a part of the smelter's toll and custom ore, in the form of refined copper cathodes (18).

In 1962 the company owned 3,034 acres at Inspiration, including the Live Oak Pit and Thornton Pit. The Christmas mine was scheduled to come into production in mid-1962. Also in 1962 the facility had a 15,500-ton-per-day capacity dual-process leaching plant and flotation concentrator at Inspiration, and a 4,000-ton-per-day capacity flotation concentrator at Christmas (16).

In 1962 the copper smelter at Inspiration had a 1,200-ton-per-day capacity furnace, a 260-ton-per-day capacity anode plant, and a 140-ton-per day capacity electrolytic refinery (16).

In 1969 mill capacities were as follows: at Inspiration, a leaching plant and flotation concentrator at 25,000 TPD; and at the Christmas mine, a flotation concentrator of 5,500 TPD (17).

In 1969 the Inspiration smelter had a 1,300 TPD furnace capacity; an anode plant of 260 TPD capacity; an electrolytic refinery of 190 TPD capacity; and a continuous-casting rod plant of 250 TPD (17).

ASARCO

Table 5 summarizes the material processed and the blister copper produced at ASARCO's copper smelter located at Hayden.

Table 5

PRODUCTION AT ASARCO'S HAYDEN SMELTER (9)

Year	Material Processed (tons)	Blister Copper Produced (tons)
1970	716,000	148,000
1971	632,000	134,000
1972	755,000	160,000
1973	746,000	147,000
1974	614,000	129,000
1975	522,000	105,000
1976	523,000	102,000

The Hayden smelter principally treats copper concentrates and precipitates produced by mines in the southwestern United States. ASARCO operates two additional smelters, with one in Texas and the other in Washington. For all three smelters combined, roughly one-third of the incoming copper is received from ASARCO's mines, approximately one-half is treated for other companies on a toll basis, and the remainder is derived from purchased materials.

To comply with pollution control standards over the past five years, the company installed a sulfuric acid plant to reduce SO_2 emissions.

Magma Copper Co.

In 1951 the Magma Copper Co. owned 2,478 acres at Superior.

In 1961 the company's Magma mill at Superior was an 1,800-ton-per-day (TPD) flotation mill, and the one at San Manuel was a 35,000 TPD flotation mill. Both locations employed a total of 3,500 workers in 1961 (16).

The Superior underground mine produces copper, gold, and silver, while the San Manuel mine produces (by underground block caving) copper, gold, silver, and molybdenite.

In 1969 the Superior smelter produced 17,618 tons of copper and the San Manuel smelter produced 95,722 tons of copper (17). In the early 1970's the Superior smelter was permanently shut down and concentrates from the mill were sent to the San Manuel smelter for processing.

Kennecott Copper Corp.

Kennecott owns and operates four open-pit copper mines in western United States. One of these, the Ray mine, is located approximately 10 miles northwest of Kearny. Molybdenum, gold, silver, and other nonferrous metals are contained in the copper ore and are extracted as by-products.

Kennecott also owns and operates a concentrating mill which converts the ore into copper concentrates by a process of crushing, grinding, and flotation. Kennecott operates a smelter at Hayden to process its copper concentrates into blister copper. The blister copper is then shipped to one of Kennecott's plants for electrorefining (i.e., at the electrorefineries at Salt Lake City, Utah or Baltimore, Maryland) or fire refining (i.e., near Silver City, New Mexico).

Two types of ore are mined at Ray: 1) a sulfide ore which is processed at the concentrating and smelting facilities located at Hayden (approximately 20 miles south of the mine), and 2) a silicate ore which is subjected to a hydrometallurgical process at a plant located adjacent to the mine site.

Waste rock removed at the mine is hauled to a waste dump and subjected to a leaching process. Water is pumped to the top of the dump and allowed to percolate downward, leaching out the soluble copper. The resulting copper-bearing solution is treated in a nearby precipitation plant to produce precipitate copper which also is shipped to the Hayden smelter.

In 1951 the company had an 8-unit concentrator-flotation plant at Hayden. At that time a large part of the mining activities were being converted from underground to open-pit operation. To maintain ore production and the treatment of 15,000 tons per day, a pit-mine crushing plant was installed at Ray and milling facilities were expanded at Hayden. At that time ASARCO's Hayden smelter was smelting the copper concentrates from Ray. The company employed 600 men in 1951 (15).

In 1969 the open-pit mine at Ray covered about 2,100 acres (17). In the same year the company completed an electrowinning plant to produce refined copper cathode from solutions from the silicate ore, vat leaching plant (design capacity 10,000 tons of ore daily) located at Rav. The company also completed a contact acid plant with a 750-ton-per-day capacity (21).

HISTORY OF OTHER INDUSTRIES IN THE STUDY AREA

Asbestos Mining and Milling

Gila County, in the vicinity of Globe, is the only area in the United States that produces high-grade, low-iron-content chrysotile asbestos. The chrysotile deposits, first discovered in this area around 1878, were not exploited until 1913. This started a rush that lasted through World War I to locate and develop deposits father northwest in the county. Additionally, this activity was extended to the eastern part of the county subsequent to the opening in 1921 of the Fort Apache and San Carlos Indian Reservations to nonmetallic mining. In 1920 annual production reached a peak of 1,200 tons, a rate not duplicated again until 1927-1928. The depression of the 1930's virtually stopped the asbestos mining through the middle of the decade. Mining increased again in the late 1930's, and by 1940 annual production reached 1,200 tons. With the exception of a few years at the end of World War II, annual production has consistently matched or exceeded this level (11).

In 1952 the General Services Administration established a depot at Globe for the purchase of strategic grades of chrysotile. This stimulated the local industry from the postwar decline, and at the peak of activity at least 16 deposits were producing asbestos or were being developed. Each year since 1962, when the purchase program was terminated, mining has been limited between two and four sites. Since 1948, annual production with few exceptions has exceeded 2,000 tons and in several years has considerably exceeded 3,000 tons (11).

Figure 1 indicates the locations of asbestos deposits in Gila County. Most of the deposits are in two areas that cover about 100 square miles in the northern part of the county, approximately 25 to 35 miles northeast of Globe.

Prior to 1941, asbestos processing mills were built at several of the mine sites. These mills were used only briefly, and all but a small portion of the material produced was actually hand-cobbed fiber. Since the post-World War II era, construction of mills has concentrated in Globe, where there are adequate power facilities (11). In the 1950's there were several mills in Globe, including those of Jaquays Mining Corporation, Metate Asbestos Corporation, and Le Tourneau Asbestos Company.

By 1970 chrysotile was being produced from only one underground mine (owned by Jaquays). From 1973 to 1974, the quantity of asbestos produced dropped 69 percent due to a loss of market caused by new EPA regulations on consumer use and handling procedures (23). As a result of these rulings and other air quality control regulations, all mills except the Jaquays facility went out of business. In 1974 the Jaquays plant, consisting of crushing, screening, and air-separation units, underwent a $100,000 modification program to comply with air pollution requirements for asbestos processing (23).

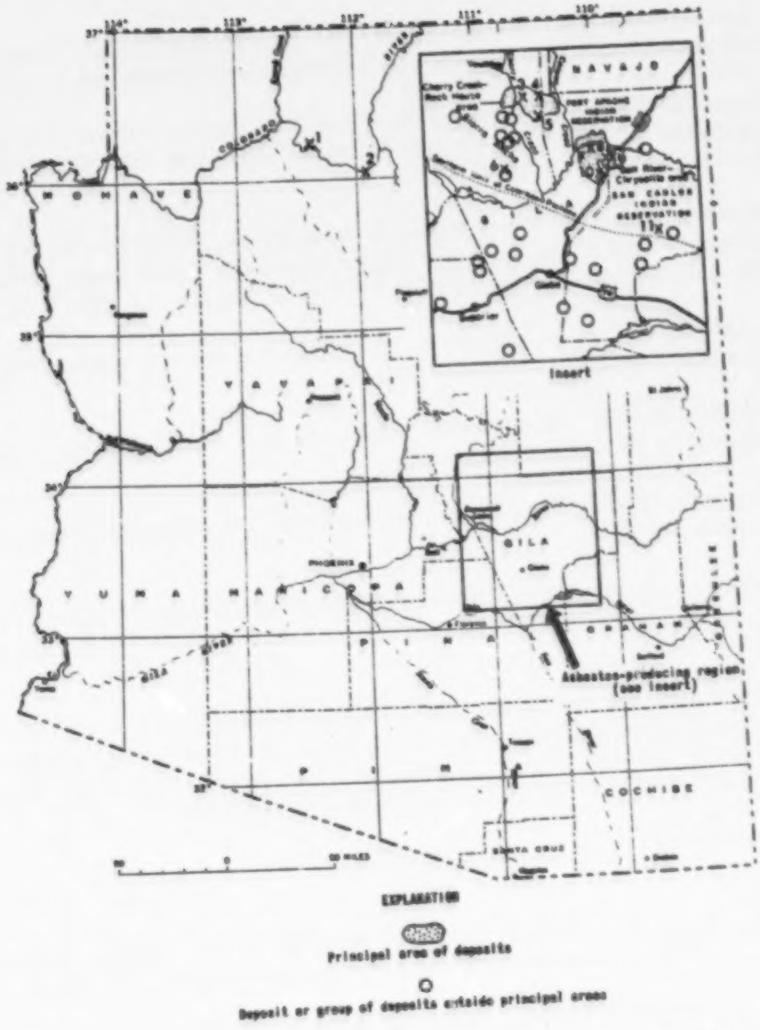
Other Mining Activities in Gila and Pinal Counties (12)

Because of the rich variety of mineral resources throughout Arizona and the study area, there are numerous types of mining activities in addition to the copper industry. Some of these other concerns are as follows:

The Arizona Gypsum Company, Pinal Mammoth Gypsum Company, and National Gypsum Company produce gypsum from open-pit mines in the lower San Pedro Valley deposits near Winkelman.

There are three open-pit mines and processing plants for crude perlite in the Miami and Superior areas. One company operates an open-pit mine, crusher, and furnace for mercury deposits in Gila County.

In addition to the copper mined by the subject industries, molybdenum, gold, and silver are also produced at the mines operated by Inspiration Consolidated Copper Company, Kennecott Copper Company, Cities Service Company (Miami Division), and Magma Copper Company's San Manuel and Superior Divisions (there is no molybdenum mining at the Superior mine).



[Source: U.S. Geological Survey, Arizona Bureau of Mines, U.S. Bureau of Reclamation, "Mineral and Water Resources of Arizona," Committee on Interior and Insular Affairs, U.S. Senate, 1969.]

Figure 1. Asbestos Deposits in Gila County

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Appendix F

MORTALITY AND POLLUTION DATA IN SUPPORT OF COPPER SMELTING CASE STUDY

Table F-1 Statistical Data for Douglas and Control
Table F-2 Statistical Data for Anaconda
Table F-3 Statistical Data for Magna and Controls
Table F-4 Data for Aggregated Arizona Smelter Towns
Table F-5 Annual Average Sulfur Dioxide Levels in Arizona Smelter Towns
Table F-6 Sulfur Dioxide Levels in Anaconda
Table F-7 Emissions from Smelter and Estimated Sulfur Dioxide Concentration for Magna, Utah
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Table F-14 Body Burden Data
Table F-15 Age-Adjusted Mortality Rates for White Females (per 100,000) for Anaconda and Controls
Table F-16 Age-Adjusted Mortality Rates for White Females (per 100,000) for Arizona Smelter Towns and Controls
Table F-17 Age-Adjusted Mortality Rates for White Females (per 100,000) for Magna and Controls
Table F-18 1976 Sulfur Dioxide Data Summary

TABLE F-1

STATISTICAL DATA FOR DOUGLAS AND CONTROL

	Observed Number of Deaths (*a*)	Relative Risk (*b*)	χ^2	Population 35-74 WF	Population All WF
Cause of Death 10					
Douglas	17			2,400	6,455
Control	978	2.1	8.6	300,341	779,118
Cause of Death 12					
Douglas	32		32.9		
Control	1,267	2.9			
Cause of Death 20					
Douglas	31		153.7		
Control	386	8.4			
Cause of Death 27					
Douglas	16		7.2		
Control	1,000	2.0			
Cause of Death 31					
Douglas	15		11.9		
Control	757	2.6			

(*a*) All ages, WF, 1968-1975.

(*b*) Note: The relative risk and χ^2 were calculated for the 35-74 age group to determine the influence of the older age group on the significance of the results. R.R. and χ^2 decreased in all cases, however, all diseases remained significantly elevated relative to the control.

Disease	R.R.	χ^2
10	2.1	6.4
12	3.2	30.1
20	5.7	40.0
27	2.0	5.5
31	3.3	16.9

TABLE F-2

STATISTICAL DATA FOR ANACONDA

	Observed Number of Deaths (*a*)	Expected Number of Deaths (*a*)	SMR	Relative Risk (*b*)	χ^2 (*c*)	Population 35-74 WF	Population All WF
Cause of Death 5							
Anaconda	17	11	160			2,188	4,984
Controls:							
Great Falls	40	47	86	2.9	7.0	10,749	30,093
Havre	8	9	86	7.9	4.3	1,766	5,079
Billings	35	48	73	6.6	5.87	11,475	31,414
Bozeman	22	13	165	0.7	0.24	2,522	9,118
Livingston	11	9	121	1.7	0.6	1,630	3,660
Cause of Death 7							
Anaconda	10	5	209				
Controls:							
Great Falls	24	21	114	1.3	0.26		
Havre	4	4	99	1.9	0.48		
Billings	16	21	75	9.5	17.9		
Bozeman	5	6	85	4.3	3.6		
Livingston	2	4	50	5.7	2.66		

TABLE F-2

(continued)

	Observed Number of Deaths *(a)*	Expected Number of Deaths *(a)*	SMR	Relative Risk *(b)*	χ^2 *(c)*
Cause of Death 19					
Anaconda	74	53	140		
Controls:					
Great Falls	245	235	104	1.5	3.0
Havre	37	51	72	2.3	4.2
Billings	243	248	100	1.8	6.0
Bozeman	72	71	101	2.3	5.4
Livingston	57	49	117	1.6	1.4
Cause of Death 27					
Anaconda	14	7	208		
Controls:					
Great Falls	36	31	117	1.7	1.6
Havre	5	6	88	4.2	3.0
Billings	39	32	122	4.2	3.2
Bozeman	5	8	61	2.1	3.7
Livingston	4	5	74	4.0	4.0

(a) All ages

(b) 35-74

(c) $\chi^2 > 3.85$ indicates $p < 0.05$

TABLE F-3

STATISTICAL DATA FOR MAGNA AND CONTROLS

	Observed Number of Deaths (*a*)	Expected Number of Deaths (*a*)	SMR	Relative Risk (*b*)	χ^2 (*c*)	Population 35-74 WF	Population All WF
Cause of Death 12							
Magna	22	18	123			1,557	4,975
Controls:							
Logan	33	43	76	1.9	4.2	2,803	11,058
Price	18	16	110	1.2	0.2	1,295	3,202
Cedar City	14	15	93	1.6	1.3	1,262	4,462
Cause of Death 14							
Magna	14	10	138				
Controls:							
Logan	21	30	70	3.5	5.4		
Price	10	10	101	3.0	2.7		
Cedar City	12	9	136	1.6	.008		
Cause of Death 18							
Magna	139	123	113				
Controls:							
Logan	353	402	88	1.8	9.8		
Price	122	124	98	1.4	2.1		
Cedar City	123	102	120	1.1	0.15		

(*a*) All ages

(*b*) 35-74

(*c*) $\chi^2 > 3.85$ indicates $p < .05$

TABLE F-4

DATA FOR AGGREGATED ARIZONA SMELTER TOWNS (Number of Deaths, White Females, All Ages, 1968-1975)

POPULATION (WF, all ages)	672	1,717	2,050	1,582	6,455	2,693	15,169	Control[a] 779,118
DISEASE CATEGORY / TOWN	Hayden	Miami	San Manuel	Morenci	Douglas	Ajo	Town Totals	Arizona
Organ Cancers:								
Cancer, oral, pharynx and esophagus	0	0	0	0	1	0		
Cancer, stomach	0	0	0	0	4	1		
Cancer, duodenum and small intestines	0	0	0	0	1	1		
Cancer, colon and rectum	0	4	0	0	13	1		
Cancer, liver and bile ducts	0	0	0	0	1	1		
Cancer, pancreas	0	2	0	2	2	0		
Other digestive cancers	0	0	0	1	0	1		
Cancer, respiratory organs	0	1	1	0	5	2		
Cancer, genitourinary organs except bladder	0	4	1	1	17	5		
Cancer, bladder	0	1	0	4	3	0		
Total	0	12	2	8	47	12	81	4,040
Other cancers except leukemia	1	7	4	5	32	7	56	1,267
Neoplasms of lymphatics	2	1	1	2	8	2	16	684
Vascular Diseases:								
Hypertension disease	0	2	0	0	4	1		
Cardiovascular diseases except stroke	12	87	11	28	206	42		
Cerebrovascular	0	0	0	7	52	13		
Total	12	89	11	35	262	56	465	25,916
Respiratory Diseases:								
Acute respiratory diseases	5	10	2	2	31	13		
Bronchitis, emphysema and asthma	0	1	0	2	6	1		
Total	5	11	2	4	37	14	76	386
Digestive Diseases:								
Other digestive diseases	0	0	0	0	0	0		
Diseases of stomach and duodenum	0	0	0	0	1	1		
Apendicitus, hernia and others	0	6	0	0	9	2		
Gastroenteritis, noninfectious	0	0	0	0	0	0		
Diseases of liver, gallbladder and pancreas	1	5	0	4	16	2		
Total	1	11	0	4	26	5	47	854
Urinary and Genital Diseases:								
Diseases of urinary system	1	6	1	1	8	1		
Diseases of the genital organs	0	0	0	0	0	0		
Total	1	6	1	1	8	1	18	587

[a] Data are from the State of Arizona minus Gila and Pinal Counties.

TABLE F-5

ANNUAL AVERAGE SULFUR DIOXIDE LEVELS IN ARIZONA SMELTER TOWNS ($\mu g/m^3$)

Location	1969	1970	1971
Ajo	98	189	50
Claypool	147	48	46
Clifton	90	135	110
Douglas ESE3	64	44	57
Douglas ENE4	109	51	55
Hayden	377	481	336
San Manuel	147	113	101
Average	147	151	107

Average for 1969-1971: 135

Source: Arizona Department of Health Services

TABLE F-6

SULFUR DIOXIDE LEVELS IN ANACONDA
(lead peroxide method)

Date of Collection	SO_2 (μg/m^3)
5/9/66 - 6/2/66	50.8
4/11/66 - 5/9/66	39.3 (low)
3/14/66 - 4/11/66	48.3
2/9/66 - 3/14/66	42.6
1/12/66 - 2/9/66	47.5
11/17/66 - 12/16/66	63.1
10/15/66 - 11/17/66	70.5
9/9/65 - 10/15/66	81.1
8/12/65 - 9/9/65	73.8
7/15/65 - 8/12/65	90.2 (high)
Average	61 ± 17

Source: Montana Air Pollution Bureau

TABLE F-7

EMISSIONS FROM SMELTER AND ESTIMATED SULFUR DIOXIDE CONCENTRATION FOR MAGNA, UTAH

Year	SO_2 Emissions Tons/Day	SO_2 ($\mu g/m^3$)
1971	193	61.8[a]
1970	261	84[b]
1969	322	103[b]

---

[a] West-Gaeke Method

[b] Values estimated by CHESS

Source: Health Consequences of Sulfur Oxides: A Report from CHESS. 1970-1971. U.S. EPA, Office of R&D, National Environmental Research Center, Research Triangle Park, N.C.

TABLE F-8

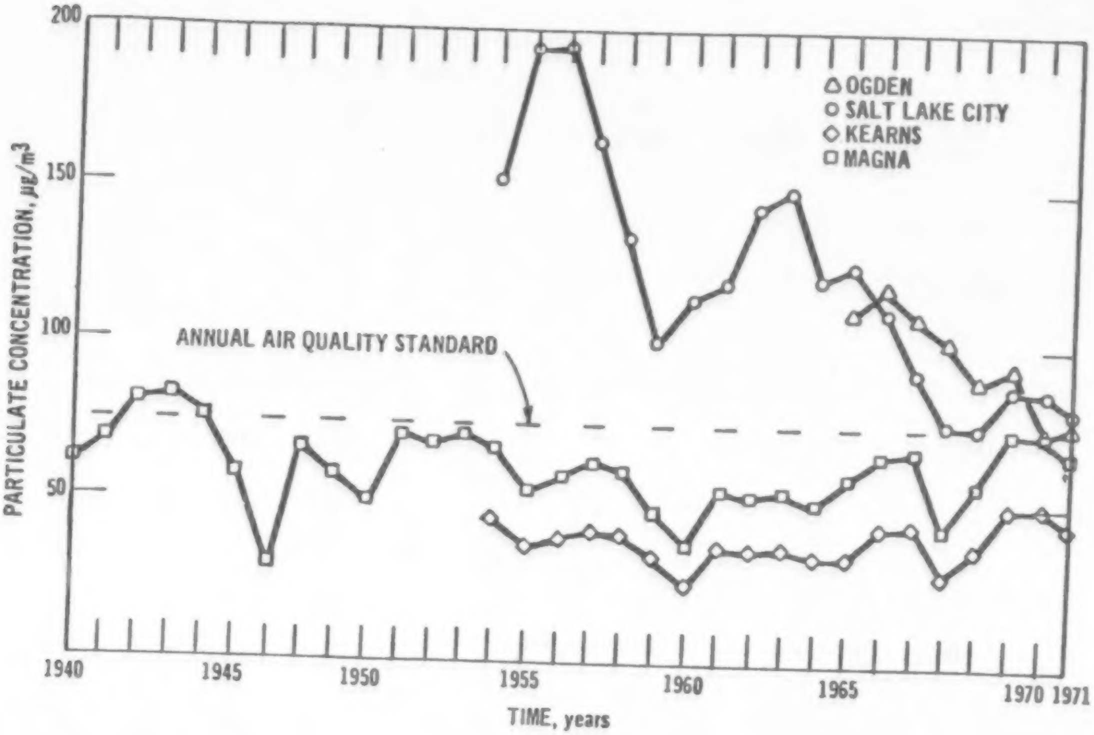
PARTICULATE LEVELS FOR ANACONDA

Year	Season	No. of Samples	Total Suspended Particulates ($\mu g/m^3$) Max.	Min.	Avg.
1961-1962	Year	123	339	10	89
1961-1962	Dec.-Feb.	30	181	11	62
1962	Mar.-May	31	339	10	115
1962	June-Aug.	32	166	51	104
1962	Sep.-Nov.	30	225	20	73

Source: Montana Air Pollution Bureau

FIGURE 1

TOTAL SUSPENDED PARTICULATE CONCENTRATIONS
VERSUS TIME FOR UTAH CHESS COMMUNITIES, 1940-1971
(Particulate Levels for Magna)



Source: Health Consequences of Sulfur Oxides: A Report from CHESS. 1970-1971. U.S. EPA, Office of R&D, National Environmental Research Center, Research Triangle Park, N.C.

TABLE F-9

SUMMARY OF PARTICULATE DATA FOR ARIZONA SMELTER TOWNS
(Annual Geometric Mean in $\mu g/m^3$)

Location	1969	1970	1971
Ajo	87	83	75
Douglas NNE	240	264	303
Hayden	224	*	*
San Manuel	43	55	*
Clifton	51	51	43
East Plantsite	51	51	*
Claypool	117	165	144
Average ± σ	116 ± 83	111 ± 86	141 ± 115

Average for
1969-1971: 122 ± 16

---

*no data available

Source: Arizona Department of Health Services

TABLE F-10

ENVIRONMENTAL CADMIUM DATA

Arizona (1969 data, $\mu g/m^3$)[a]

Smelter Towns	
Ajo	.005
Claypool	.02
Douglas	.011
E. Plantsite	.006
Hayden	.321
San Manuel	.002
Average ± σ	.006 ± .005

Non-Smelter Towns	
Davis Dam	.002
Florence	.013
Organ Pipe	.002
Page	.002
Average ± σ	.004 ± .005

Anaconda (1971-1972 data, $\mu g/m^3$)[b]

American Junior High School	.02
Kirkeby Residina	.02
Farmers Union	0
S&N Cement	.06
Johnson's Residence	.04
Bailey's Residence	.04
Average ± σ	.03 ± .02

Soil Analysis[c]: Cd (ppm) = 3.6

Magna (Nov. 1970 - Feb. 1972 data, mg/m^2/month)[d]

Nov. '70	0.17	May	-	Nov.	0.07
Dec.	0.00	June	0.14	Dec.	0.04
Jan. '71	0.05	July	0.05	Jan. '72	0.05
Feb.	0.00	Aug.	0.07	Feb.	0.02
March	0.19	Sept.	0.16		
April	0.20	Oct.	0.23		

1971 Average ± σ - .11 ± .08

Comment: There are no environmental standards for cadmium. The 1963 National Air Sampling Network found a range of 0.004 to 0.026 $\mu g/m^3$ in non-urban areas. The highest value in the survey was 0.050 $\mu g/m^3$ in Covington, Kentucky. Standard soil level is < 1 ppm.

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[a] Arizona Department of Health Services.

[b] Montana Air Pollution Bureau.

[c] Sample taken by Enviro Control site visit team, November 1977.

[d] CHESS study (This data is the only cadmium data for Magna found; it is in units not directly comparable to the Arizona and Montana data.)

TABLE F-11

ENVIRONMENTAL LEAD DATA

Arizona (1969 data, μg/m^3)[a]

Smelter Towns	
Ajo	.1
Claypool	1.2
Douglas	.5
E. Plantsite	2.2
Hayden	3.2
San Manuel	.04
Average ± σ	1.2 ± 1.2

Non-Smelter Towns	
Davis Dam	.1
Florence	.5
Organ Pipe	.1
Page	.04
Average ± σ	.18 ± .2

Anaconda (1961-1962 data, μg/m^3)[b]

Period		No. of Samples	Max.	Min.	Avg.
1961-1962	Dec.-Feb.	30	1.7	.2	.70
1962	Mar.-May	31	1.7	.1	.53
1962	June-Aug.	32	2.1	0	.48
1962	Sep.-Nov.	30	3.2	.03	.46
1961-1962	Year	123	3.2	0	.54

Magna (Nov. 1970 - Feb. 1972 data, mg/m^2/mo.)[c]

Nov. '70	22.98	May	-	Nov.	2.41
Dec.	1.84	June	4.96	Dec.	2.02
Jan. '71	3.21	July	1.26	Jan. '72	2.30
Feb.	1.95	Aug.	5.46	Feb.	2.53
March	7.75	Sept.	4.02		
April	6.08	Oct.	4.82		

Average ± σ = 3.89 ± 2.2

Comment: There are no environmental standards for lead. The National Air Sampling Network reported a range of .002 to .15 μg/m of lead in non-urban areas. The highest in the survey was 2.7 μg/m in Minneapolis.

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[a] Arizona Department of Health Services

[b] Montana Air Pollution Bureau

[c] CHESS study (This data is the only lead data for Magna found. It is in units which are not directly comparable to the Arizona and Montana data.)

TABLE F-12

ENVIRONMENTAL ARSENIC DATA[a]

Arizona (1969 data, $\mu g/m^3$)[b]

Smelter Towns	
Ajo	.003
Claypool	.01
Douglas	.004
E. Plantsite	.007
Hayden	.006
San Manuel	.009
Average ± σ	.0065 ± .0027

Non-Smelter Towns	
Davis Dam	.001
Florence	.01
Organ Pipe	.005
Page	.001
Average ± σ	.0042 ± .0042

Anaconda (1961-1962 data, $\mu g/m^3$)[c]

Period		No. of Samples	Max.	Min.	Avg.
1961-1962	Dec.-Feb.	30	2.5	0	.54
1962	Mar.-May	31	1	0	.3
1962	June-Aug.	32	1.9	0	.52
1962	Sep.-Nov.	30	1.8	0	.5
1961-1962	Year	123	2.5	0	.45

Soil Analysis[d]: November 1977, 33 ppm

Comment: There is no ambient standard for arsenic. However, a study by the Montana State Board of Health found that the value for Anaconda was higher than any other city in the state as well as New York and Chicago. Normal soil levels are about 8 or 9 ppm.

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[a] No data available on Magna.

[b] Arizona Department of Health Services.

[c] Montana Air Pollution Bureau.

[d] Sample taken by Enviro Control site visit team, November 1977.

TABLE F-13

FLUORIDE CONTENT OF RANDOM GRASS SAMPLES
(Deer Lodge Valley)

Location	Date of Collection	Concentration in ppm	Date of Collection	Concentration in ppm
Anaconda (west)	7/15/65	12.6		
Anaconda (north)	11/3/65	3.7		
Opportunity (east)	11/3/65	5.0		
Anaconda (east)	11/3/65	3.7		
Deer Lodge (east)	8/19/65	2.2		
Galen (west)	8/19/65	2.0	6/30/66	4
Deer Lodge (west)	8/11/65	2.0	6/30/66	2
Galen (east)	8/19/65	3.2		

Source: Montana Air Pollution Bureau

TABLE F-14

BODY BURDEN DATA

Heavy Metal Levels in Hair, Blood and Urine[a]

- Mean ± S. E. (number of samples) -

Location		Hair Lead (μg/gm)	Blood Lead (μg/100 ml)	Blood Electrocyte Protoporphyrin (μg/100 ml)	Urine Arsenic (μg/l)	Hair Arsenic (μg/gm)	Hair Cadmium (μg/gm)
Smelter Towns	Ajo	18.9 ± 1.48 (85)	12.5 ± .46 (105)	22 ± .9 (105)	C0.8 (77)	1.9 ± .14 (85)	26.8 ± .22 (85)
	Anaconda	25.5 ± 1.6 (86)	13 ± .9 (64)	26 ± 1.7 (64)	30.8 (40)	19.8 ± 1.9 (86)	1.8 ± 1.6 (86)
	Douglas	25.9 ± 1.8 (95)	20 ± .8 (97)	30 ± 1.9 (97)	18 (80)	.43 ± .02 (95)	1.72 ± .15 (95)
	Hayden	26 ± 1.5 (104)	21 ± .8 (100)	30 ± 1.8 (100)	27 (33)	2.01 ± .14 (104)	1.78 ± .12 (104)
	Miami	31.6 ± 2 (78)	17 ± .7 (94)	27 ± 1.6 (94)	15 (72)	.47 ± .05 (78)	1.00 ± .16 (78)
	Morenci	18 ± 1.3 (98)	13 ± .5 (100)	23 ± 1.3 (100)	15 (77)	.31 ± .02 (78)	1.9 ± .13 (98)
	San Manuel	14 ± 2.7 (14)	18 ± .5 (101)	23 ± 1.1 (101)	22 (55)	.18 ± .04 (80)	0.90 ± .21 (14)
Comparison Towns	Albuquerque	17 ± 2 (22)	17 ± .6 (81)	26 ± 1.4 (82)	6.9 (51)	.09 ± .01 (22)	.41 ± .08 (22)
	Perryville MO	12 ± 1 (86)	16 ± .7 (85)	21 ± .9 (86)	3.9 (81)	.08 ± .01 (86)	1.04 ± .13 (86)
	Safford AZ	11 ± .9 (52)	15 ± .7 (92)	23 ± 1.2 (92)	8.2 (61)	.1 ± .01 (52)	.96 ± .14 (52)

Note: Underscored towns are significantly different from comparison towns.

Summary of Arithmetic Means for Hair Samples, by City and Metal (ppm)[b]

	Pb	Cd	As
Bozeman	7.6	.9	.41
Anaconda	14.3	1.3	10.6

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[a] Baker, E.L., Carl G. Hayes, Philip J. Landrigan, Jan L. Handke, Ronald T. Leger, W. Jere Housworth, and J. Malcom Harrington. (1977) A nationwide survey of heavy metal absorption in children living near primary copper, lead and zinc smelters. *American Journal of Epidemiology* 106(4):261-273.

[b] Data from Ecological Research Branch, EPA. (Pre-report) Fall 1969.

TABLE F-15

AGE-ADJUSTED MORTALITY RATES FOR WHITE FEMALES (per 100,000) FOR ANACONDA AND CONTROLS

CAUSE OF DEATH (ECI #)	AGE-ADJUSTED MORTALITY RATES					
	Anaconda	Controls				
		Great Falls	Havre	Billings	Bozeman	Livingston
Infectious & parasitic diseases (1)	0	4	0	3	9	0
Cancer, oral, pharynx & esophagus (2)	0	11	0	8	0	0
Cancer, stomach (3)	6	7	8	5	4	0
Cancer, duodenum & small intestine (4)	0	0	0	0	0	0
Cancer, colon & rectum (5)	56	19	14	21	74	30
Cancer, liver & bile ducts (6)	0	8	0	7	0	0
Cancer, pancreas (7)	39	21	21	7	10	8
Other digestive cancers (8)	0	0	0	0	0	0
Cancer, respiratory organs (9)	27	30	27	20	9	21
Cancer, genitourinary organs except bladder (10)	45	45	30	37	31	32
Cancer of the bladder (11)	0	1	0	1	4	8
Other cancers except leukemia (12)	98	100	84	71	96	38
Neoplasms of lymphatics (13)	20	21	34	18	18	61
Endocrine, nutritional, metabolic & blood disease (14)	24	34	7	21	20	67
Mental & nervous system diseases (15)	18	20	7	14	8	17
Rheumatic heart disease (16)	29	15	20	20	14	41
Hypertensive heart disease (17)	0	8	7	8	4	14
Cardiovascular diseases (18)	443	308	253	280	228	338
Cerebrovascular diseases (19)	133	86	55	74	58	88
Respiratory diseases (20)	45	23	34	31	22	58
Bronchitis, emphysema & asthma (21)	19	19	0	21	10	33
Pneumoconiosis (22)	0	0	0	0	0	0
Other digestive diseases (23)	8	0	0	0	4	0
Diseases of stomach & duodenum (24)	10	3	0	2	4	0
Appendicitis, hernia & others (25)	24	9	14	14	0	0
Gastroenteritis, noninfectious (26)	0	0	0	0	0	0
Diseases of liver, gallbladder & pancreas (27)	56	35	13	27	14	15
Diseases of urinary system (28)	11	10	7	6	9	0
Diseases of the genital organs (29)	0	1	0	1	0	0
Diseases of pregnancy & childbirth (30)	0	0	0	0	0	0
Diseases of the skin & musculoskeleton & ill-defined diseases (31)	21	66	20	42	14	15
Congenital anomalies (32)	0	3	0	3	5	0
Perinatal diseases (33)	0	0	0	0	0	0
Accidents, poisons & violence (34)	25	53	43	58	63	32

TABLE F-16

AGE-ADJUSTED MORTALITY RATES FOR WHITE FEMALES (per 100,000) FOR ARIZONA SMELTER TOWNS AND CONTROLS

CAUSE OF DEATH (ECI #)	AGE-ADJUSTED MORTALITY RATES						
	Arizona Smelter Towns						Controls
	Miami	Hayden	San Manuel	Morenci	Douglas	Ajo	
Infectious & parasitic diseases (1)	0	0	0	0	11	13	75
Cancer, oral, pharynx & esophagus (2)	0	0	0	0	5	0	33
Cancer, stomach (3)	0	0	0	0	15	12	7
Cancer, duodenum & small intestine (4)	0	0	0	0	0	16	0
Cancer, colon & rectum (5)	0	0	0	0	33	0	29
Cancer, liver & bile ducts (6)	0	0	0	157	6	0	2
Cancer, pancreas (7)	0	0	0	0	5	0	12
Other digestive cancers (8)	0	0	0	0	0	13	0
Cancer, respiratory organs (9)	23	0	72	12	15	26	33
Cancer, genitourinary organs except bladder (10)	56	0	0	43	63	58	31
Cancer of the bladder (11)	0	0	0	0	0	0	0
Other cancers except leukemia (12)	131	102	95	86	110	49	35
Neoplasms of lymphatics (13)	23	41	0	46	20	30	17
Endocrine, nutritional, metabolic & blood disease (14)	17	102	0	78	57	29	19
Mental & nervous system diseases (15)	0	0	0	0	17	0	10
Rheumatic heart disease (16)	62	0	36	0	9	0	12
Hypertensive disease (17)	18	0	0	0	10	16	1
Cardiovascular diseases (18)	910	529	314	487	382	239	323
Cerebrovascular diseases (19)	0	0	0	187	79	62	77
Respiratory diseases (20)	57	188	0	31	60	69	10
Bronchitis, emphysema & asthma (21)	18	0	0	0	19	16	0
Pneumoconiosis (22)	0	0	0	0	0	0	0
Other digestive diseases (23)	0	0	0	0	0	0	0
Diseases of stomach & duodenum (24)	0	0	0	0	5	0	6
Appendicitis, hernia & others (25)	36	0	0	0	29	12	12
Gastroenteritis, noninfectious (26)	0	0	0	0	0	0	0
Diseases of liver, gallbladder & pancreas (27)	62	85	0	25	67	24	33
Diseases of urinary system (28)	18	0	0	31	15	0	9
Diseases of the genital organs (29)	0	0	0	0	0	0	1
Diseases of pregnancy & child-birth (30)	0	0	0	0	0	0	0
Diseases of the skin & musculoskeleton & ill-defined diseases (31)	17	84	15	16	61	0	18
Congenital anomalies (32)	0	0	0	0	0	11	2
Perinatal diseases (33)	0	0	0	0	0	0	0
Accidents, poisons & violence (34)	143	53	35	94	61	38	45

TABLE F-17

AGE-ADJUSTED MORTALITY RATES FOR WHITE FEMALES (per 100,000) FOR MAGNA AND CONTROLS

CAUSES OF DEATH (ECI #)	AGE-ADJUSTED MORTALITY RATES			
	Magna	Controls		
		Logan	Price	Cedar City
Infectious & parasitic diseases (1)	5	0	9	15
Cancer, oral, pharynx & esophagus (2)	9	0	0	0
Cancer, stomach (3)	0	7	0	0
Cancer, duodenum & small intestine (4)	0	0	0	0
Cancer, colon & rectum (5)	33	13	20	30
Cancer, liver & bile ducts (6)	0	5	6	0
Cancer, pancreas (7)	0	10	23	7
Other digestive cancers (8)	0	0	0	0
Cancer, respiratory organs (9)	0	12	16	7
Cancer, genitourinary organs except bladder (10)	54	9	12	29
Cancer of the bladder (11)	0	0	0	0
Other cancers except leukemia (12)	131	63	98	75
Neoplasms of lymphatics (13)	15	12	15	23
Endocrine, nutritional, metabolic & blood disease (14)	77	24	26	59
Mental & nervous system diseases (15)	21	16	26	23
Rheumatic heart disease (16)	25	42	35	44
Hypertensive disease (17)	9	10	26	15
Cardiovascular diseases (18)	379	206	265	322
Cerebrovascular diseases (19)	70	70	52	60
Respiratory diseases (20)	11	23	38	22
Bronchitis, emphysema & asthma (21)	0	4	13	8
Pneumoconiosis (22)	0	0	0	0
Other digestive diseases (23)	0	0	0	0
Diseases of stomach & duodenum (24)	20	2	0	15
Appendicitis, hernia & others (25)	15	11	6	0
Gastroenteritis, noninfectious (26)	0	0	0	0
Diseases of liver, gallbladder & pancreas (27)	33	21	29	15
Diseases of urinary system (28)	20	10	0	7
Diseases of the genital organs (29)	0	0	0	0
Diseases of pregnancy & child-birth (30)	0	0	0	0
Diseases of the skin & musculoskeleton & ill-defined diseases (31)	57	11	14	22
Congenital abnormalities (32)	10	0	0	8
Perinatal diseases (33)	0	0	0	0
Accidents, poisons & violence (34)	43	41	50	45

TABLE F-18

1976 SULFUR DIOXIDE DATA SUMMARY (in μg/m^3)

Site	Location	Operator	Method	1976 Annual Average
Smelter Towns:				
Ajo	Town Square, approx. 0.5 mi, N54°W from smelter stack	PD	Coul	12
Ajo	Oxidation Pond, approx. 1.2 mi, N8°E from smelter stack	PD	Coul	33
Ajo	S. Tailings Dam, approx. 1.3 mi, N85°E from smelter stack	PD	Coul	11
Ajo	Camelback Mtn., approx. 1.2 mi, N88°E from smelter stack	PD	Coul	7
Ajo	AZ DOT Well Rd.	State	Coul	21
Average ±σ				16 ±10
Douglas	Northwest, approx. 1.22 mi, N30°W of smelter stack	PD	Coul	51
Douglas	Pirtleville, 1.3 mi, N22.5°E of smelter stack	PD	Coul	43
Douglas	2 km NNW of stack	State	Coul	43
Douglas	U.S. 666, 2 km NNE of stack	State	Coul	39
Douglas	North, 0.75 mi, N15°E of smelter stack	PD	Coul	39
Douglas	Curtis, approx. 2.65 mi, N32°W of smelter stack	PD	Coul	31
Douglas	Fairgrounds, approx. 3 mi, N55°E of smelter stack	PD	Coul	17
Douglas	Fir, approx. 14 mi, N47°E of smelter stack	PD	Coul	33
Douglas	1012 G. Avenue (Mercantile)	PD	Coul	31
Douglas	Martin, approx. 4.2 mi, N20°E of smelter stack	PD	Coul	14
Average ±σ				34 ±11
Hayden	Approx. 1 mi North of the junction of Highway 77 and 177	JCC	Coul	135
Hayden	Hayden Fire Station, 640 Canyon Drive	JCC	Coul	105
Hayden	Hayden Jct. - Highway 177	JCC	Coul	57
Hayden	Montgomery Ranch	State	Coul	127
Hayden	Montgomery Ranch	JCC	Coul	183
Hayden	Old Town Jail - Canyon Drive	State	Coul	113
Average ±σ				103 ±29
Miami	Jones Ranch	State	Coul	56
Miami	Lower Miami School	State	Coul	21
Average ±σ				38 ±24
Morenci	Cadillac Point - 5500, 1.75 mi, N51°W of smelter stack	PD	Coul	105
Morenci	Clifton Fina, 1.75 mi, S69°E of smelter stack	PD	Coul	34
Morenci	East Plantsite, 2.5 mi, S35°E of smelter stack	PD	Coul	7
Morenci	Metcalf, approx. 3.9 mi, N28°W from smelter stack	PD	Coul	83
Morenci	Oroville, approx. 2.75 mi, N48°E of smelter stack	PD	Coul	9
Morenci	Stargo, 1.5 mi, N63°W of smelter stack	PD	Coul	109
Morenci	Eagle Creek Road, approx. 4.75 mi, S84°W of smelter stack	PD	Coul	16
Morenci	Fairbanks, approx. 2.1 mi, S26°E of smelter stack	PD	Coul	13
Morenci	Stargo, Highway 666	State	Coul	130
Morenci	U.S. 666, 4.5 km NW of stack	State	Coul	108
Average ±σ				49 ±52

(continued)

Source: 1976 Air Quality Data for Arizona, reported by the Arizona Department of Health Services

TABLE F-18 (continued)

Site	Location	Operator	Method	1976 Annual Average
Smelter Towns (continued)				
San Manuel	Upper Shopping Center, 2 km SSW of smelter stacks	Magma	Coul	45
San Manuel	Golf Course, 3 km WNW of smelter stacks	Magma	Coul	75
San Manuel	East Peppersauce Wash, 5.8 km ESE of smelter stacks	Magma	Coul	17
San Manuel	LDS Church	State	Coul	42
San Manuel	Mine site, 10.5 km NW of smelter stacks	Magma	Coul	42
Average ±σ				44 ±20
Nonsmelter Cities				
Phoenix	1845 E. Roosevelt	Maricopa	Coul	7
Phoenix	241 N. Central, Valley Bank Center, 29th Floor	PD	Coul	8
Phoenix	15 E. Monroe, Valley Bank Annex, 3rd Floor	PD	Coul	8
Phoenix	1740 W. Adams	State	Bubbler	7
Phoenix	1845 E. Roosevelt	State	Bubbler	7
Average ±σ				7 ±.5
Tucson	32 N. Stone, Home Federal Tower, 6th Floor	PD	Coul	7
Tucson	151 W. Contress	Pima	Flame	7
Tucson	Florence Hwy/McGee Rd.	Pima	Coul	8
Tucson	Sahuaro Monument	Pima	Coul	4
Tucson	1721 N. Tanque Verde Loop	Pima	Coul	6
Tucson	22nd/Craycroft	Pima	Coul	5
Tucson	University of Arizona	State	Bubbler	6
Average ±σ				5 ±2

Appendix G

SELECTED DISEASE CATEGORIES AND INCLUDED ICDA CODES FOR COPPER SMELTING CASE STUDY

Table G-1

SELECTED DISEASE CATEGORIES AND INCLUDED ICDA CODES

ECI Disease Number	Disease Group	ICDA Codes (8th Rev.)	
5	Cancer of colon and rectum	153,154	Cancer of colon and rectum
7	Cancer of pancreas	157	Cancer of pancreas
10	Cancer of genitourinary organs except bladder	180	Cancer of cervix uteri
		181	Chorionepithelioma
		182	Other malignant neoplasms of uterus
		183	Cancer of ovary, fallopian tube and broad ligament
		184	Cancer of other and unspecified female genital organs
		185	Cancer of prostate
		186	Cancer of testis
		187	Cancer of other and unspecified male genital organs
12	Other cancers except leukemia	170-174	Malignant neoplasm of bone
		190-199	Cancer of other and unspecified sites (eye, brain, thyroid, and other endocrine gland, lymph nodes in head, face and neck, secondary neoplasms)
		210-239	Benign neoplasms of digestive and respiratory systems, bone, skin, connective tissue, musculature, genitourinary organs, nervous and endocrine systems
14	Endocrine, nutritional, metabolic, and blood diseases	240-289	Diseases of thyroid gland; other endocrine glands (pituitary, adrenal, gonadal dysfunction); avitaminoses and other nutritional deficiency; other metabolic diseases (congenital disorders of amino-acid, carbohydrate and lipid metabolism); other diseases such as plasma protein, porphyrin, etc., disorders; diseases of blood and blood-forming organs
18	Vascular diseases	410-458	Ischemic heart disease (myocardial infarction, chronic ischemic heart disease with and without hypertensive disease); diseases of pericardium, endocardium, and myocardium; pulmonary heart disease; congestive heart failure; disorders of cardiac rhythm; cerebrovascular diseases (including cerebral hemorrhage, embolism and ischemia); arteriosclerosis; phlebitis
19	Cerebrovascular diseases	430-438	Hemorrhage, with and without hypertension; thrombosis; ischemia
20	Acute respiratory diseases	460-486	Acute nasopharyngitis, acute bronchitis and bronchiolitis; influenza; pneumonia
		500-508	Chronic pharyngitis, sinusitis; larynx disorders
		510-519	Pleurisy; pneumothorax; pulmonary congestion and hypostasis; pneumoconiosis; interstitial pneumonia; bronchiectasis; pulmonary collapse; edema of the lung; emphysema
27	Diseases of liver, gallbladder and pancreas	570	Acute and subacute necrosis of liver
		571	Cirrhosis of liver
		571.0	Alcoholic cirrhosis of liver
		572	Suppurative hepatitis and liver abscess
		573	Other diseases of liver
		574	Cholelithiasis
		576	Other diseases of gallbladder and biliary ducts
		577	Diseases of pancreas
31	Disease of skin and musculo-skeletal system and ill-defined diseases	680-739	Boils and carbuncles, cellulitis, acute lymphadenitis, skin infections; other inflammatory conditions of skin and subcutaneous tissue; other diseases of skin and subcutaneous tissue (disease of sweat and sebaceous glands)
		780-796	Arthritis and rheumatism, other bone and joint diseases (osteomyelitis, etc.); diseases of the musculoskeletal system (myositis, myasthenia gravis); connective tissue disorders; congenital disorders of the nervous system (spina bifida, hydrocephalus); circulatory, respiratory, gastrointestinal, urogenital, chromosomal abnormalities; perinatal diseases; disorders of pregnancy; birth injuries, anorexia; prematurity

Appendix H

DISEASE ASSOCIATIONS IN AMERICAN STEEL MINING COMMUNITIES

Appendix H

DISEASE ASSOCIATIONS IN AMERICAN STEEL MILL COMMUNITIES*

There is little direct information on disease risk among the resients of communities that are economically dominated by steel mills. What information exists is concerned almost exclusively with cancer mortality. One must therefore extrapolate from the known emission and effluent products of this industry and from occupational data.

CHARACTERISTICS OF STEEL COMMUNITIES

In general, steel cities are less stereotyped in their cultural and ethnic profile than coal mining communities. They may be discreet communities such as Bethlehem, Pennsylvania; metropolitan areas such as Gary, Indiana; or districts of a much larger metropolis such as the Sparrow's Point/Dundalk area of Baltimore, Maryland. They often have large ethnic minorities; this vastly complicates interpretation of certain diseases rates, such as cancer of the stomach, which are thought to be strongly influenced by dietary or cultural factors. Steel communities are often on the receiving end of large population migrations, especially from Appalachia and the deep South, so that a subset of the population may reflect the risk profile of a distant, very different locale. In short, steel communities are not as simple in their demographic and epidemiologic characteristics as other industrial cities may be.

HAZARDS ASSOCIATED WITH STEEL MILL OPERATION

The hazards associated with blast furnace operation (as outlined in Volume II of the Plan for Investigation) require some medical comment:

- *Gaseous Emissions from the Blast Furnace*—Although oxides of nitrogen are undoubtedly present as a consequence of the high temperatures achieved in blast furnaces, none of the communities cited by the Environmental Protection Agency as exceeding ambient air quality criteria for NO_x in the period 1971-1973 were steel cities except for Baltimore, where there is also a large concentration of other industries. On the other hand, it is quite plausible that oxides of sulfur are significant as a health hazard in these communities, with the associated risk of chronic bronchitis and chronic obstructive pulmonary disease.

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*This report was prepared for Enviro Control, Inc., by Tee L. Guidotti, M.D., medical consultant on this project.

- *Particulate Matter*—It is not entirely clear what agents may exist in the ash of blast furnaces. The ash of coke ovens, however, is of great concern due to its content of potential carcinogens; there may be 1% benzo(a)pyrene (BaP) in such particles.

- *Leachates Into the Water Table*—These may carry a variety of metal contaminants, including arsenic, lead, and cadmium. Water used for cooling purposes, on the other hand, is likely to be fairly free of toxic metals.

- *Gaseous Emissions from Metal Processing; Flue Dust Fines after Cyclone Separation; Metal Fines from Furnace Processes*—This group is much more likely to pose an occupational threat to workers than to be a risk to the outside population.

These hazards are unique to the blast furnace. Hazards associated with other basic steel processes include:

- *Coke Oven Fumes*—As will be shown, these constitute a major occupational hazard. There is as yet no clear evidence that they are a risk beyond the plant gates, but this probably depends on the local siting of the coke ovens. These emissions are prime candidates for implication in any community cancer excess.

- *Silicates from Firebrick*—These particles from crubling firebrick may aerosolize in the respiratory range and are the cause of silicosis in workers at the blast furnace. They are unlikely to be implicated in community risk.

- *Asbestos*—This is used in heat shields and in various uses around the plant. Its ubiquity makes it an important factor in assessing cancer risk. Furthermore, it is already demonstrated to be significant in conjugal and community exposure.

- *Heat*—The intense heat generated by the blast furnace may be more than an occupational hazard. It may be significant in atmospheric reactions which change the composition of air pollution (analogous to its role in producing the oxides of nitrogen). Furthermore, it may volatilize metal or other normally particulate compounds and render them airborne.

- *Machine Oils*—There is a well-documented excess of lung and oropharyngeal cancer among machinists, and there is emerging evidence that the agent may be the fine petroleum oils with which the piece and the machinery are lubricated. This may be a factor in finishing operations; it is unlikely to be significant in basic steel production.

DISEASES OF STEELWORKERS

By and large, steelworkers tend to be healthy and have overall cause-specific mortality rates substantially lower than the general population. Their causes of death are parallel to those of the general population, except that they have a disproportionate rate of accidents.

A large and elegant body of work has been accumulated on cancer mortality among steelworkers. It is now well established that workers in the coke ovens, who constitute a small fraction of steelworkers as a whole, are at enormously high risk for lung cancer. This association is exposure- and cumulative-dose-related. There is a lesser excess of cancer of the rectum. There are highly suggestive, but not definitive, associations for cancer of the kidney (hypernephroma—an uncommon but not rare tumor) and bladder, and a possible association with cancer of the pancreas, in workers associated with other, mostly finishing processes. This pattern of organ involvement strongly suggests a carcinogen which (1) is inhaled, (2) is excreted by the kidney and concentrated in urine, and (3) may be incidentally ingested as well. Thus, there may be more to an excess risk of cancer among steelworkers than lung cancer in coke oven workers—and it should still be emphasized that steelworkers in general have a lower mortality from cancer at all sites than the general population.

Metalworkers who work with steel, but who are not necessarily steelworkers, show an interesting pattern of cancer mortality in Britain. They have a significant excess mortality from lung cancer and questionable excesses for thyroid and bone cancers. The latter two are relatively unusual in the general population, and one wonders whether there might be a carcinogenic agent which is handled metabolically in much the same way as either iodine or calcium. Since the mortality rates for cancers at all sites (aggregated) are lower among steelworkers than in the population at large, rates for cancers at specific sites which do not achieve statistical significance for excess when compared to the general population may be strikingly more significant if compared to an occupational control cohort.

Heart disease as a cause of death was seen in excess only in certain finishing operations and among janitors and general laborers (whites only—blacks did not share this excess risk). Nonetheless, maintenance workers, who also sustain the intermittent, variable exposure which is similar to that suffered by the community at large, had a remarkably low mortality from heart disease. Cerebrosvascular disease and stroke were elevated to significance in one finishing operation, but not in basic steel processes. An exhaustive study of cardiovascular disease and myocardial infarction among British steelworkers operating the blast furnaces could document no excess risk, despite elevated blood levels of carbon monoxide.

DISEASE PATTERNS IN STEEL COMMUNITIES

There is little information on disease incidence in steel communities beyond cancer mortality.

The wives of boilermakers, an occupation which involves steel fabricating but not exposure to machine oils, have a slightly increased mortality from cancer at all sites (aggregate) in Britain and, specifically, elevated mortality from lung cancer. Although the standardized mortality ratio is not striking (132), there may be a factor at work in conjugal exposure or in overall elevated community risk.

County cancer mortality for those parts of the country that have concentrations of steel mills disproportionate to other concentrations of industry, such as Pittsburgh, Birmingham, and Gary, show elevated rates at all sites (aggregate) for both males and females, further suggesting a community risk. Cancer of the stomach and of the lung and, interestingly, of the rectum appear at higher risk in both sexes in Pittsburgh (Allegheny County). Cancer of the oropharynx (mouth and throat), larynx, and esophagus—all uncommon and very likely related to environmental exposure—are at excess in men only (corrected for the known male predominance of these tumors anyway). This suggests but cannot prove a local occupational exposure. As pointed out in Volume II of the Plan for Investigation, cancer rates in general were found to be in excess for residents of a Canadian steel city, Hamilton, and attempts have been made to associate this with fluoride emissions. On a biological basis, this is not particularly convincing, although it is possible that fluorides are a marker for some other biologically active pollutant which is released in a similar manner.

The following, therefore, emerge as the diseases most likely to be found in excess in steel communities:

- Cancer of the lung
- Cancer of the stomach
- Cancer of the rectum
- Cancer of the pancreas
- Cancer of the bladder
- Cancer of the kidney (hypernephroma)
- Cancer of the thyroid (it would be useful to identify the tissue type)
- Chronic bronchitis and chronic obstructive pulmonary disease
- Upper and lower respiratory tract infections (unlikely to be a major cause of death, however)
- Cancer of the head and neck

There is at present no evidence for an excess of cardiovascular disease, although one cannot truly generalize from basically health steelworkers to the effect on an entire population.

Several important disease processes are overlooked in current studies. The opportunity exists to complete this omission by supplying the missing elements in the profile of steel community health. These include:

- Diseases of the liver and biliary tract (bearing in mind that high rates of alcoholic cirrhosis may exist)
- Congenital malformations
- Spontaneous abortions and miscarriages

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Appendix I

AERIAL PHOTO ANALYSIS PERFORMED BY ENVIRONMENTAL PHOTOGRAPHIC INTERPRETATION COMPLEX (EPIC)

Appendix I

AERIAL PHOTO ANALYSIS PERFORMED BY ENVIRONMENTAL PHOTOGRAPHIC INTERPRETATION COMPLEX (EPIC)

Methodology for Johnstown and Lewistown, Pennsylvania Air Photo Analyses

EPIC's primary tasks performed for this project included (1) the identification of changes in residential patterns in the Johnstown area from 1939 to 1972; (2) the identification and location of significant point and non-point sources of pollution in the area; (3) the analysis of the development of industry in the area from 1939 to the present. In addition, an estimate was made (for Johnstown only) of the area containing the highest ground level concentration of air pollution (principally sulfur dioxide and particulate matter) based upon a simple "box" diffusion model.

Aerial photography was used as the data source for most of the required work, and black and white photography dating back to 1939, at a scale of 1:24,000, was utilized in the interpretation and analysis process. Some recent (1977) color aerial coverage was also used in analyzing industrial activity in the area. For the concentration estimates, emissions and meteorological data were obtained from Enviro Control and EPA/Research Triangle Park, North Carolina. Additional data on diffusion estimates for the Johnstown area is yet to be received from TRP.

The finished product consisted of several 7.5 minute USGS map sheets with acetate overlays showing residential changes, point and non-point sources, and the estimated area of maximum ground level concentration. A written text was prepared describing industrial activity in the area from 1939 to the present with respect to changes in size, new construction, and addition of air pollution control equipment. In addition, a photo-mosaic with an overlay depicting the principal industries was also prepared.

An almost identical study was also done for the Lewistown, Pennsylvania area. In this instance, however, an air pollution concentration and area estimation was not undertaken, and the only aerial photography used was from the years 1938 to 1964. Table I-1 delineates the aerial photo coverage and the overlays prepared for Johnstown and Lewistown, Pennsylvania.

Table I-1

DATES OF AERIAL PHOTO COVERAGE FOR JOHNSTOWN AND LEWISTOWN, PENNSYLVANIA

GEOGRAPHICAL AREA		DATE				
		Topography Map	Overlay			Photo-Revision
			#1	#2	#3	
LEWISTOWN	Lewistown	1966	1938	1964	--	1973
	Burnham	1966	1938	1964	--	
JOHNSTOWN	Johnstown	1964	1938	1962	1972	--
	Geistown	1964	1938	1962	1972	1972
	Hooversville	1971	1938	1962	1972	--
	Windber	1971	1938	1962	1972	--
	Nanty Glo	1964	1938	1962	--	1972
	Vintondale	1964	1938	1962	1972	1972

Industrial Activity and Development in the Johnstown, Pennsylvania Area

(see Figure I-1)

Site A

This medium-sized industrial plant is engaged in heavy fabrication, probably structural steel, and was built between 1939 and 1955. It is very probably associated with Bethlehem Steel or U.S.Steel. There is only one medium-sized stack associated with the plant, and no plume is visible from it. The plant has only expanded once, by about 30%, between 1972 and 1977. No air pollution control equipment is presently attached to the stack.

Site B

This site is actually composed of two brick kiln complexes located directly across the river from each other. Both of these were built prior to 1939. One kiln had been removed from the west bank complex between 1939 and 1955. Between 1939 and 1955, three kilns had been added, and other facilities expanded at the east bank complex. All of the kilns on the west bank had been removed between 1962 and 1972, and all operations had been terminated. The complex on the east bank appears to have gone out of operation during this time period also, and all of the facilities were physically removed between 1972 and 1977.

Site C

This large fabricating (probably structural steel) plant is associated with Bethlehem Steel, and was built before 1939. There are two small stacks, and several very small stacks or "chimneys" on the roof,which have been in place since before 1939. No stack plumes, or air pollution control equipment, are visible on 1977 aerial photography. The plant was expanded by approximately 20% between 1939 and 1955, and water clarifiers or mixing tanks were installed between 1972 and 1977. Other than that, no changes have taken place since 1955.

Site D

Two complexes (Da and Db) comprise this large Bethlehem Steel operation. Complex Da is engaged in coke production, and complex Db is engaged in coke and coke by-products prodution, and heavy fabrication (probably structural steel). In 1939 there were four medium-sized stacks serving Da, and three medium-sized stacks serving Db. Portions of the fabricating plants in complex Db were rebuilt between 1955 and 1962, and one medium-sized stack had been removed. There has been no change in the number of coke ovens at either

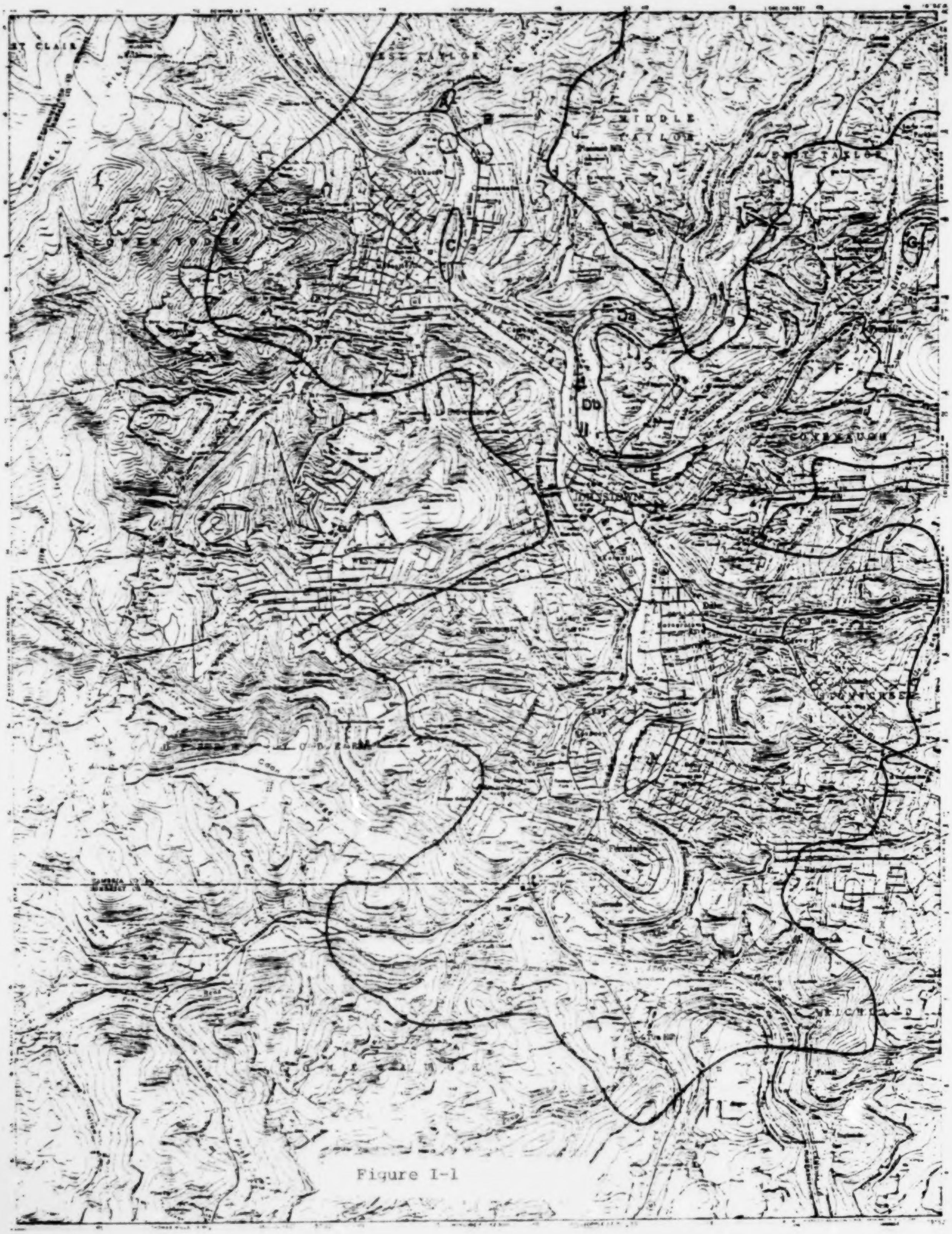
ST CLAIR
WEST TAYLOR
MIDDLE TAYLOR
EAST TAYLOR
LOWER YODER
A
B
C
DD
F
G
CONEMAUGH
STONYCREEK
RICHLAND

Figure I-1

complex since 1939. Another medium-sized stack was removed from Db between 1962 and 1972. Two medium-sized stacks were removed from, and two small stacks were added to the Da complex between 1962 and 1972. No stack plumes or air pollution control equipment were visible on 1977 aerial photography.

Site E

This large Bethlehem Steel complex appears to be engaged in structural steel fabrication, and was built prior to 1939. Approximately ten small stacks were visible at the site in 1939. Several of the smaller stacks had been removed and replaced with two medium-sized stacks between 1962 and 1972. No other changes have taken place since 1939, and no stack plumes or air pollution control equipment were visible on 1977 aerial photography.

Site F

This very large complex is the Franklin Works (iron smelter and steel plants), which is owned and operated by Bethlehem Steel Corporation. It has many small and medium-sized stacks, and contributes approximately 80% of the ambient sulfur dioxide and particulate matter loading in the Johnstown area. Some open hearth furnaces had been removed, and some blast furnaces had been added between 1955 and 1962. Some other plant facilities were also removed between 1962 and 1972. Electrostatic precipitators were installed between 1972 and 1977 to handle many of the dust-producing processes.

Site G

This Bethlehem Steel plant appears to be engaged in structural steel fabrication. It was built prior to 1939, and has only one small stack. The plant expanded approximately 10% between 1939 and 1955, and by another 10% between 1962 and 1972.

Site H

This site is a small light industrial plant, with one medium-sized stack, built before 1939. It was replaced by an office building between 1962 and 1972.

Site I

This stack is associated with a small power plant for a junior high school.

Site J

U.S.Steel owns and operates this heavy fabrication/structural steel complex. It was built prior to 1939, and has approximately 6 or 7 small stacks. One plant in the complex was rebuilt between 1962 and 1972. Three of the taller stacks appear to have some type of pollution control equipment installed on them.

Site K

The stack is for the heating plant connected to a complex of greenhouses.

Site L

The stack is also for a heating plant connected to a complex of greenhouses.

Site M

The medium-sized stack depicted here is for a small power plant associated with the State Rehabilitation Center.

Site N

The stack annotated here is medium-sized, and associated with a small industrial power plant.

Johnstown, Pennsylvania Concentration Estimates

- Must assume uniform distribution in the valley (area delineated on map; see Figure I-1)
- Average valley width in the delineated area is approx. 12,000 ft or 3658 m
- Mixing depth is approximately 550 ft or 168 m

Pollutant Emissions:

	Normal	Peak
Total Sulfur Dioxide	423 g/sec	667 g/sec
Total Particulate Matter	212 g/sec	217 g/sec

$$\chi = \frac{Q}{u} \left(\frac{1}{D}\right) \left(\frac{1}{y}\right)$$

χ = Ground Level Concentration

Q = Total Emissions

u = Average Wind Speed

D = Mixing Depth

y = Average Valley Width

SO_2 Concentration Estimates

$$\chi = \frac{4.23 \times 10^{8}\ \mu g/sec}{6\ m/sec} \left(\frac{1}{3658\ m}\right) \left(\frac{1}{168\ m}\right)$$

$$\chi = (7.05 \times 10^{7})(2.7 \times 10^{-4})(6.0 \times 10^{-3})$$

χ = 11.4 × 10 or 114 $\mu g/m^3$ (normal)

$$\chi = \frac{6.67 \times 10^{8}}{6} \left(\frac{1}{3658}\right) \left(\frac{1}{168}\right)$$

χ = 180 $\mu g/m^3$ (peak)

Particulate Matter Estimates

χ = 57 $\mu g/m^3$ (normal)

χ = 58.2 $\mu g/m^3$ (peak)

Appendix J

MORTALITY RATE COMPUTATION METHODOLOGY
and
ECI, SSI AND ICDA CAUSE OF DEATH CODE CONVERSION TABLES

Appendix J

MORTALITY RATE COMPUTATION METHODOLOGY
AND
ECI, SSI AND ICDA CAUSE OF DEATH CODE CONVERSION TABLES

FOR SITES OF INTEREST

1. Print the number of deaths, populations, age-specific mortality rates, and age-adjusted mortality rates for these places as follows:

a. White males; white females; nonwhite males; nonwhite females

b. Age groups:
0- 2
3-14
15-24
25-34
35-44
45-54
55-64
65-74
75 and over

Note: Age-adjusted mortality rates to be calculated using data from 35 to 74 year age groups only.

c. Causes of death:

ECI CAUSE-OF-DEATH CODES
COMPARED TO 8TH REVISION (1968 AND AFTER) ICDA CODES

ECI #	ICDA Code (8th Revision)	Cause of Death
1	000-136	Infectious & parasitic diseases
2	140-150	Cancer, oral, pharynx & esophagus
3	151	Cancer, stomach
4	152	Cancer, duodenum & small intestine
5	153-154	Cancer, colon & rectum
6	155-156	Cancer, liver & bile ducts
7	157	Cancer, pancreas
8	158-159	Other digestive cancers
9	160-163	Cancer, respiratory organs
10	180-187,189	Cancer, genitourinary organs except bladder
11	188	Cancer of the bladder
12	170-174,190-199,210-239	Other cancers except leukemia
13	200-209	Neoplasms of lymphatics
14	240-289	Endocrine, nutritional, metabolic & blood diseases
15	290-380	Mental & nervous system diseases
16	390-398	Rheumatic heart disease
17	400-404	Hypertensive disease
18	410-458	Cardiovascular diseases
19	430-438	Cerebrovascular diseases
20	460-486,500-508,510-514,516-519	Respiratory diseases
21	490-493	Bronchitis, emphysema & asthma
22	515	Pneumoconiosis
23	520-530	Other digestive diseases
24	531-537	Diseases of stomach & duodenum
25	540-560,562-569	Appendicitis, hernia & others
26	561	Gastroenteritis, noninfectious
27	570-577	Diseases of liver, gallbladder & pancreas
28	580-599	Diseases of urinary system
29	600-629	Diseases of genital organs
30	630-678	Diseases of pregnancy & childbirth
31	680-739,780-796	Diseases of skin & musculoskeleton & ill-defined diseases
32	740-759	Congenital anomalies
33	760-779	Perinatal diseases
34	800-1000	Accidents, poisons & violence

ECI CAUSE-OF-DEATH CODES
COMPARED TO 7TH REVISION (BEFORE 1968) ICDA CODES

ECI #	ICDA Code (7th Revision)
1	000-138,696-697,764,767-769
2	140-150
3	151
4	152
5	153-154
6	155
7	157
8	158-159
9	160-164
10	171-180
11	181
12	156,165,170,190-199,210-219,222-239
13	200-205
14	250-299,772
15	300-326,340-390,765
16	400-416
17	441-447
18	330-334,420-434,450-468
19	330-334
20	470-500,510-527,763
21	240-241,501-502,527
22	523
23	530-539
24	540-545
25	550-570,572-578
26	571
27	580-587
28	590-609
29	610-637
30	640-689
31	221,242-244,690-695,698-749,766,780-795
32	220,750-759
33	760-762,769-776
34	800-1000

CAUSE-OF-DEATH CODES FOR CANCER REGISTRY DATA

ECI#	ICD #'s	Site of Neoplasm
C-1	400-509	Oral, pharynx, esophagus
C-2	510-519	Stomach
C-3	520-529	Small intestine
C-4	530-539	Colon
C-5	540-549	Rectum
C-6	550-559	Liver
C-7	560-579	Gallbladder, pancreas
C-8	580-599	Other digestive
C-9	600-619	Nasal, larynx
C-10	620-629	Trachea, bronchus, lung
C-11	630-659	Other respiratory and intrathoracic organs
C-12	690-699	Hematopoietic and reticuloendothelial systems
C-13	700-739	Bone, connective tissue, skin
C-14	740-759	Breast
C-15	790-899	Genitourinary
C-16	880-889	Bladder
C-17	900-929	Eye, nervous system
C-18	930-949	Endocrine glands
C-19	950-959	Ill-defined
C-20	960-969	Lymph nodes
C-21	990-999	Unknown

SSI CAUSE-OF-DEATH CODES COMPARED TO 8TH REVISION ICDA CODES

SSI Code	ICDA Code (8th Revision)	Disease Categories
1	000-E999	All deaths
2	000-136,290-389,630-679,710-739,760-E999	Infective and parasitic diseases; mental disorders; diseases of the nervous system and sense organs; complications of pregnancy, childbirth and the puerperium; diseases of the musculoskeletal system and connective tissue; certain causes of mortality in early infancy; symptoms and ill-defined conditions; and accidents, poisonings and violence
3	140-289,390-629,680-709,740-759	All other deaths, including neoplasms; endocrine, nutritional and metabolic diseases; diseases of the blood and bloodforming organs; diseases of the circulatory system; diseases of the respiratory system; diseases of the digestive system; diseases of the genitourinary system; diseases of the skin and subcutaneous tissue; congenital anomalies
4	140-239	All neoplasms
5	140-149	Buccal cavity and pharynx
6	150-159	Digestive organs and peritoneum
7	151	Stomach
8	152-153	Small and large intestines
9	154	Rectum
10	157	Pancreas
11	150,155-156,158-159	Esophagus, liver, gallbladder, peritoneum and other digestive organs
12	160-163	Respiratory system
13	162	Trachea, bronchus and lung
14	160-161,163	Other parts of the respiratory system
15	174	Breast
16	180-184	Female genital organs
17	185-187	Male genital organs
18	188	Bladder
19	189	Other urinary organs
20	191-192	Brain and other parts of the nervous system
21	204-207	Leukemia
22	200-203,208,209	Other neoplasms of lymphatic and hematopoietic tissues
23	170-173,190,193-199,210-239	Other malignant neoplasms and benign neoplasms
24	250	Diabetes mellitus
25	240-246,251-279	Other endocrine, nutritional and metabolic diseases
26	280-289	Diseases of blood and bloodforming organs

(continued)

SSI CAUSE-OF-DEATH CODES COMPARED TO 8TH REVISION ICDA CODES
(concluded)

SSI Code	ICDA Code (8th Revision)	Disease Categories
27	390-448	Major cardiovascular disease
28	390-398	Chronic rheumatic heart disease and active rheumatic fever
29	400-404	Hypertensive disease
30	410-414	Ischemic heart disease
31	410-411	Acute myocardial infarction and other acute or subacute forms of ischemic heart disease
32	412-414	Chronic ischemic heart disease and angina pectoris
33	420-429	Other forms of heart disease
34	430-438	Cerebrovascular disease
35	440	Arteriosclerosis
36	441-448	Other diseases of arteries, arterioles and capillaries
37	450-458	Diseases of veins, lymphatics and other diseases of circulatory system
38	460-519	Diseases of the respiratory system
39	484-485	Acute interstitial and bronchopneumonia
40	490-493	Bronchitis, emphysema and asthma
41	490-491	Bronchitis
42	292	Emphysema
43	493	Asthma
44	510-511,513,517	Empyema, pleurisy, and other chronic interstitial pneumonia
45	460-483,486,500-508,512,514-516,518,519	Other diseases of the respiratory system
46	520-577	Diseases of the digestive system
47	571	Cirrhosis of liver
48	571.0	Alcoholic cirrhosis of liver
49	571.8-571.9	Other and unspecified cirrhosis of liver
50	520-570,572-577	Other diseases of the digestive system
51	580-629	Diseases of the genitourinary system
52	580-584	Nephritis and nephrosis
53	590	Infections of kidney
54	591-629	Other diseases of the genitourinary system
55	680-709	Diseases of the skin and subcutaneous tissue
56	741-759	Congenital anomalies

2. Calculations:

a. Age-adjusted mortality rates using only the 35-74 year age groups (see note on page J-1) for each place or aggregate:

$$A(t) = \sum_{i=1}^{n} \frac{S(i,t)\,P(i)}{\sum_{i=1}^{n} P(i)}$$

where:

$S(i,t\ )$ = the age-specific death rate in age group i over t years

$$S(i,t) = \frac{D(i,t)}{P(i)} \times \frac{100{,}000}{8}$$

$A(t)$ = the age-adjusted death rate over t years

$D(i,t)$ = the number of deaths in age group i over t years

$P(i)$ = the number in age group i in the U.S. standard population

t = the number of years observed

i = a particular age group

n = the number of age groups

b. U.S. standard population:

Age	Number	%
35-44	20,327,701	29.35
45-54	20,846,499	30.10
55-64	16,811,212	24.27
65-74	11,281,137	16.29

3. Population Data: 1970 fifth count, file C, census tapes for sites of interest.

4. Mortality Data: Death certificate tape for sites of interest.

5. Summary Demographic Statistics

For places of interest, summary demographic statistics from 1970 census tapes as follows:

median age
median income
% in same house in 1965
% white
% ≤1.00 persons per room
% finishing high school
% female
% native of native parents
% electricity used for heating

Appendix K1

ETIOLOGY OF CARDIOVASCULAR DISEASES ASSOCIATED WITH BITUMINOUS COAL MINING

Appendix K2

ETIOLOGY OF RESPIRATORY DISEASES ASSOCIATED WITH BITUMINOUS COAL MINING

Appendix K1

ETIOLOGY OF CARDIOVASCULAR DISEASES ASSOCIATED WITH BITUMINOUS COAL MINING*

The incidence data for cardiovascular disease in coal mining communities is misleading in several respects when one attempts to generalize risk data to a broader population than miners alone. The greatest source of error is the intrinsic interrelation of pulmonary and cardiovascular disease, such that most patients with advanced pulmonary disease will as a matter of course develop heart disease. Another source of confusion is the absence of data on uncomplicated atherosclerotic cardiovascular disease alone in coal mining communities, so that no population model exists for estimating risk in coal regions in general. Finally, miners are predisposed by their working conditions to develop peripheral venous disease (thrombophlebitis, varicose veins), and this should not be allowed to complicate consideration of arterial disease.

HEART DISEASE IN COAL MINERS

Although there appears to be a moderate elevation in mortality from "heart disease" in general and "myocardial infarction" in particular, these data must be interpreted very cautiously. An excess mortality from "heart disease" is a predictable result of a high prevalence of lung disease, because the pulmonary and cardiovascular systems are intimately related. Mechanisms for this interdependence are primarily the following:

a) Progressive destruction of lung tissue also reduces the cross-sectional area of the pulmonary arterial bed. Reduced oxygen tensions also induce vascular constriction in the pulmonary artery. These phenomena result in reduced compliance, therefore increased resistance of the pulmonary arterial bed, which is normally a very low resistance hydrodynamic system. This increased resistance puts a markedly increased work load on the right heart; over time, the right ventricle hypertrophies (increases its muscle mass) to accommodate the need to generate excessive pressure. If the right ventricle cannot sustain the additional work, it will fail, a condition known as "right heart failure" which has several distinct features from the more common left heart failure. In addition, the hypertrophic right ventricle may demand more oxygen than its overextended coronary arteries are capable of supplying, producing angina pectoris. The phenomenon of right ventricular hypertrophy in association with lung disease is known as "cor pulmonale" and has been well documented to occur in coal miners with complicated coal workers' pneumoconiosis.

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*This report was prepared for Enviro Control, Inc., by Tee L. Guidotti, M.D., medical consultant on this project.

b) Distension of the right atrium by the same mechanism described above and hypoxemia due to poor oxygen exchange by severe lung disease sensitize the heart to arrhythmias. In severe acid/base disturbances, which are not uncommon in chronic lung disease, the heart is also made more irritable. Foci in the cardiac conduction system and myocardium are thus more susceptible to spontaneous depolarization and may escape from electrical domination by the sinus node. Thus, irregularities in rhythm are among the most common consequences of moderate to severe lung disease. At the extreme, these arrhythmia can be fatal.

c) Severe lung disease is a chronic process of symptomatic improvement followed by exacerbation in a stepwise downhill course. It is rarely a smooth history of decline, as cardiac disease more commonly may be. An intercurrent illness, such as influenza, may be all that is needed to acutely decompensate the patient and precipitate a cardiac problem. Unless a physician is attuned to this in his record-keeping, the interrelation may be lost in chart review.

d) When a patient ceases to breathe as a result of lung disease and its attendant disturbance in gas exchange, the next event to occur (after brain damage, which does not declare itself immediately) is cardiac arrest. Undoubtedly, many deaths from lung disease are recorded as deaths from cardiac arrest.

It should also be noted that primary cardiac disease can have dramatic effects on the lungs, in the form of pulmonary edema, congestive heart failure, pulmonary emboli, hemoptysis, and so forth. As a general rule, however, these are less likely to be mistaken for primary lung disease in records review.

The incidence of cardiac complications of lung disease in coal mining communities is not the primary question of a disease risk survey, although it may be interesting from the standpoint of clinical management. The fundamental problem is the risk of lung disease per se and the risk of primary cardiac disease per se.

Of overriding importance in assessing disease risk is the question of predisposition to atherosclerotic coronary artery disease. Data on aggregate "heart disease" does not help to approach this problem because of the reservations outlined above. One study that looked at the incidence of peripheral arterial disease in another arterial bed (cerebral) is described below and does not suggest any particular predisposition.

ARTERIAL DISEASE IN COAL MINERS

Stroke is the result of a disturbance in the arterial blood supply to the brain. By far the most common causes are complications of atherosclerosis and hypertension. (Congenital deformities are much less common, usually occur in younger patients, and tend to be clinically distinguishable. Traumatic causes of brain infarction are not considered "strokes.") Hypertension and atherosclerosis are closely associated, and it is generally not useful to separate them for epidemiological purposes. The incidence of stroke can be assumed to reasonably reflect the incidence of severe peripheral vascular disease in general.

The best information on the incidence of stroke in coal mining communities comes from a study of coal miners conducted in 1967 by the United Mine Workers in an attempt to audit their health services. The population consisted of bituminous coal miners in Western Pennsylvania, and the diagnostic criteria used were clinical and comparable to other studies. They derived an incidence of 7.9 strokes per 1,000 program-eligible miners per year, a figure comparable to the population at large in several other studies. There is thus no evidence for an increased incidence of stroke among bituminous coal miners.

It would appear unlikely that coal miners are significantly predisposed to peripheral arterial disease and, by extension, such a predisposition would be even more unlikely in the nonmining population of coal mining communities.

VENOUS DISEASE IN COAL MINERS

This is briefly discussed in Appendix K2. The basic disease process of concern is venous distension and thrombosis caused by sluggish blood flow. Coal miners sit or crouch for long periods in awkward positions, or may stand for long periods at the face without moving around very much. These are well known to cause stasis of venous flow, and the high incidence of venous disease in miners comes as no surprise. When superficial veins dilate as a result, they become typical varicose veins; however cosmetically undesirable and troublesome to the patient, they are trivial as a cause of death. Much more serious is in situ thrombosis and thrombophlebitis. The clot which forms on the wall of the vein may break off and travel in the venous circulation to the lung (through the right heart); this is pulmonary embolism, which is a major cause of death from vascular disease. Pulmonary embolism usually results from thrombophlebitis of the deep veins of the legs, especially in men.

It is highly unlikely that venous disease in coal miners has anything to do with coal dust exposure; the high incidence is easily explicable by the working conditions in the mines. There is no reason to believe that there is any increased risk whatsoever in nonminer residents of mining communities.

Portal vein thrombosis is a very serious, rare complication of cirrhosis and occasionally of infections involving organs that drain into the portal vein. The excess in coal miners probably simply represents the high prevalence of cirrhosis. There is very little reason to believe that it has anything to do with coal dust exposure, and it is so infrequent as to be unrewarding in search.

There is no reason to suspect any elevation in risk for peripheral venous disease in nonminer residents of coal mining communities.

CARDIOVASCULAR DISEASE WHICH MIGHT BE FOUND IN EXCESS IN COAL-MINING COMMUNITIES

ICDA numbers 410 (myocardial infarction), 411 (acute/subacute ischemic heart disease), 412 (chronic ischemic heart disease), and 413 (angina pectoris) are different manifestations of the same basic disease process: atherosclerotic coronary artery disease. If coal mining community residents are predisposed to accelerated atherosclerosis, these diseases should be uniformly elevated in risk. This data is easily confused by the high prevalence of lung disease. If chart review data is to be used to derive a risk estimate, *every effort must be made to exclude cardiac complications of lung disease*. The etiology of such complications is fundamentally different than that of primary cardiac disease, and lung disease will in effect be counted twice if it cannot be separated out. The best available data, though limited, suggests that mining populations have no particular predisposition to accelerated atherosclerosis.

ICDA number 414 (asymptomatic ischemic heart disease) is not particularly useful in view of the high frequency of relatively trivial abnormalities on electrocardiogram; the chart data available will not be sufficient to support the diagnosis consistently from patient to patient.

ICDA numbers 450 (pulmonary embolism), 451 (thrombophlebitis), 452 (portal vein thrombosis), 453 (other venous disease), 454 (lower extremity varices), and 456 (varices, other sites) have satisfactory alternative explanations. The chance that a community population risk could be found is exceedingly small.

ICDA number 457 (noninfective disease of lymphatic channels) is most likely a reflection of edema and probably overlaps with 454. There is no reason to suspect any relationship between coal and any form of extrapulmonary lymphatic disease. (Lymphoma, malignancy of the lymphatic system, is coded under other designations.)

ICDA number 458 (other diseases of the cardiovascular system) is essentially so vague as to be useless and undoubtedly overlaps with the more specific codes cited above.

CONCLUSIONS

Searching for an elevated risk from cardiovascular disease in coal mining communities will probably be low in yield. If any excess were found, it would most likely reflect cardiac complications of primary pulmonary disease. It is possible, but unlikely, that atherosclerotic coronary artery disease could be increased in risk; it would appear in several ICDA classifications and should also be reflected in incidence data for stroke. Venous disease and lymphatic disease are so unlikely to be elevated in community risk that they may be essentially ignored.

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Appendix K2

ETIOLOGY OF RESPIRATORY DISEASES ASSOCIATED WITH BITUMINOUS COAL MINING*

1. DISEASE INCIDENCE IN AMERICAN BITUMINOUS COAL MINING COMMUNITIES

Studies of disease incidence in coal mining communities have concentrated on the coal miner himself. There has been little attention paid to the nonminer residents of the regions where coal is mined, and what studies exist come mostly from the United Kingdom. One finds a dearth of information on the effect of this industry on community health in the United States.

Characteristics of American Coal Mining Communities

Coal-mining communities in the United States differ from other settlements in several ways which may affect epidemiologic trends.

a) Active bituminous coal mining in the United States is concentrated in one geographic region, Appalachia, which has had a high prevalence of infectious pulmonary disease. Historically, tuberculosis has been relatively more prevalent and, until recently, less well controlled than in other regions. Histoplasmosis is endemic to the area as well.

b) Coal mining communities, especially in Appalachia, have been subject to wildly fluctuating economic circumstances, which have led to a skewed age distribution in the population. Some Appalachian counties lost virtually their entire healthy male cohort aged 20 to 40 during the peak years of emigration in the 1950's. Thus, the age structure of individual communities in Appalachia may confuse estimates of the incidence of age-related, especially chronic, diseases. There may also be a bias toward overrepresenting chronic disease presenting in youth or in the complications of institutionalization or mental deficiency, since such conditions will occur disproportionately in the adult male population which did not emigrate, as the result of selection pressures.

c) Poverty and inadequate health services, especially in the most rural areas of Appalachia, may affect the quality of incidence data, since serious disease may be underreported. Depending on the continuity of local medical care, diagnostic data may not be accurate in the distant past.

d) Coal mining communities tend to be relatively homogeneous in socioeconomic class and ethnic origin (there are certain important exceptions to this).

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*This report was prepared for Enviro Control, Inc., by Tee L. Guidotti, M.D., medical consultant on this project.

Diseases of Bituminous Coal Miners

Coal miners themselves are predisposed to a variety of illnesses, chiefly respiratory. There are four major occupational respiratory diseases of coal miners:

- Coal Workers' Pneumoconiosis, simple (CWP); this results from the accumulation of inhaled coal dust in discrete macules in the lung and can be associated with some changes in respiratory function, rarely if ever disabling.
- Progressive Massive Fibrosis (PMF, or "complicated CWP); this is an advanced, uncommon form of CWP which results in obstructive (emphysematous) and restrictive pulmonary disease and may be life threatening.
- Chronic Bronchitis; this probably represents chronic bronchial irritation from dust, but has not been extensively studied.
- Silicosis; this occurs in miners who drill rock, sink shafts, or drive the transport cars ("motormen") and can be life threatening in severity.

Lung cancer in coal miners is a controversial issue. American coal miners have been reported to have excess mortality from lung cancer, but this has not been found in the United Kingdom. (The British data may be diluted by other job categories.) Furthermore, coal miners have been reported to have excess cancer of the upper respiratory tract (pharynx, possibly larynx), which is much less frequent than cancer of the lung. If there were a weak carcinogenic substance in coal dust, there might be an elevated community risk for cancer of the lung and upper respiratory tract in settlements with heavy dust fallout or in the families of miners. It is therefore interesting to note that there is indeed an excess of cancer of the oropharynx in coal mining counties in Appalachia, but not of lung cancer.

Tuberculosis is reportly more prevalent in U.S. coal miners than in all U.S. working men, but again this has not been found in the U.K. One must recall that TB was poorly controlled in Appalachia in general until recently, so that a comparison with all U.S. working men is not satisfactory.

Miners in general have excess mortality from pneumonia and from pulmonary embolism. The latter surely reflects their prolonged awkward posture underground, which predisposes to venous stasis and deep vein thrombosis.

Nonrespiratory diseases to which coal miners appear to be at excess risk include cancer of the stomach, gastric ulcer, hepatic cirrhosis, and syphilis. The last three are perhaps better explained by factors of lifestyle than by occupation. Miners also have high rates of fatal and disabling accidents on the job, and of homicide.

Disease Patterns in Bituminous Coal Mining Communities

There is data quoted in earlier work on this project to suggest increased respiratory disease in nonminer residents of coal mining communities, but this did not prove consistent between communities. In Appalachia there is a shared risk of tuberculosis and of histoplasmosis complicating these findings.

Relatively few cancers seem to cluster in Appalachian coal mining counties; these include cancer of the cervix, upper respiratory trace (oropharynx), skin (all types), and bone (all types). One intriguing association from the U.K. suggests excess mortality from leukemia among the wives of coal miners.

The disease experience of coal miners which may plausibly result from an exposure shared by nonminer residents of the community can be added to the scant data available on coal mining communities themselves to yield a list of conditions most likely to be found in excess in such settlements.

The following emerge as the most likely diseases to be found in excess in bituminous coal mining communities:

- Chronic Obstructive Pulmonary Disease (emphysema, chronic bronchitis)
- Tuberculosis
- Cancer of the Lung
- Cancer of the Head and Neck
- Pneumonia
- Skin Cancer
- Leukemia

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from the bedrock to where the coal seam lies; e.g., various mines in one area with a deep seam may be called "Peabody No. 9" or "Oak Creek No. 11."

Strip mining is an efficient method of extracting coal but is suitable only for more shallow seams and larger operations; it carries a substantial additional cost for reclamation of the land stripped off the seam and replaced—the "overburden." Occasionally, strip and underground mines coexist in the same coalfield. Underground mines are constructed by first drilling a vertical shaft, then a horizontal corridor. Perpendicular to the corridor, shallow "panels" are cut at intervals. The panels are enlarged into "rooms" when the exposed "face" of the seam is to be "cut," or mined, at that point. The floor plan of a mine is therefore a rectilinear array of rooms coming off a main corridor and numerous accessory corridors, branching from the main corridor. A few rooms, having served their purpose in mining, are turned into crude workshops or garages for machinery. This room-and-pillar arrangement may be the structurally safest and most efficient floor plan* but still leaves 65% of the coal unmined between the rooms, serving as pillars to support the roof.

Access to a mine is through a "portal," usually an electric elevator in the vertical shaft. Electric shuttlecars run by "motormen" on rails negotiate the main corridor. In the past, sand was sprinkled on the steel rails to provide traction; this resulted in clouds of airborne pulverized silica and led to a high incidence of silicosis among motormen and brakemen.

At the face of the seam, an operative with a motorized drilling machine drills about 10 holes, each 10 feet deep, into the face and inserts two sticks of dynamite in each. Electronic detonation is triggered by a "shotfireman" after the nearby rooms are evacuated; the area is reentered about five minutes later, after the fumes from the explosion have been allowed to dissipate. The atmosphere is tested with a lantern burning a low flame, called a "canary" for reasons of historical whimsy. Although acrid and smoky, the area is normally thought safe to reenter shortly. In the past, this process known as "shotfiring" exposed many miners to toxic concentrations of nitrogen dioxide when they reentered prematurely (4).

The next step is "undercutting," in which a six-ton machine with a cutting tool like that of a chain saw cuts a slot about 12 cm high, two meters wide, and over a meter deep into the base of the seam, which is usually 50 to 70 cm high in Appalachia. The largest machine in the mines, the continuous miner, then chews into the seam and conveys the broken chunks of coal into a bin, where it is loaded into a loading machine for transport. These operations at the face of the mine are extremely dusty, and these workers sustain the heaviest exposure to coal dust** (5).

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*An alternative plan, called "longwalling," is the predominant system in Europe and is used by some American mines.

**Masks are very seldom worn, as they are very uncomfortable, hot, and impair vision.

The coal is brought to a conveyor belt, a V-shaped belt travelling about a meter per second through a small accessory shaft to the surface, carrying loads of coal 15 to 20 cm deep. Although against safety regulations, it is quite common for men to hitch a fast ride to the surface by jumping on the conveyor belt. At the surface, the coal is dumped in a "main pit" where it is soon loaded onto another conveyor belt, the "slope belt" which lifts it high in the air and deposits it on the top of a huge pile, the "tipple." The coal is stored there until it is loaded onto trucks or railroad cars, by being lifted again by conveyor belt into a huge funnel, the "cyclone." The mechanics and the surface workers, who are often older underground miners retiring from work at the face, are considered active "miners" but have much less exposure to coal dust than other workers.

As the face is cut and the coal is removed, the room is deepened by a cutting machine. This is a potentially very hazardous operation, since the integrity of the ceiling or "roof" cannot be guaranteed. A specialized operative called the "roofbolter" drills dozens of 2-meter holes perpendicularly into the roof and secures it with long reinforcement bolts with expandible heads and plates. Drilling into solid rock exposes the roofbolter, like the shaft driller on the construction crew, to the risk of silicosis.

Roof falls are the most common fatal accidents in underground mining, so that great care is taken to reinforce the corridors with oak or steel beams, to roofbolt the rooms, and the sound the roof for "kettle bottoms." These are large, semicylindrical petrified tree trunk fragments lying over the seam. (When the coal was still a swamp, these would have been floating or mired logs.) They may drop precipitously when undercut and are exceedingly dangerous.

Each underground miner carries his light on his helmet wired to a battery on his belt, his survival pack, and his lunch pail. They work in teams of nine, with a foreman, mechanic (or electrician), motorman, two roofbolters, and operatives for each machine. An underground miner's average workday in the mine proper is only 6.5 hours; the rest of his time is spent in transit and setting up the machinery.

Sources of first-hand information on coal-mining operations are not limited to industry and union representatives. Trade journals such as *Coal Age* and *Coal Mining and Processing*, publications of the U.S. Bureau of Mines and the U.S. Mine Enforcement and Safety Administration (MESA), and university departments of engineering are helpful, once one becomes familiar with the jargon. Although operating mines discourage visitors, there are exhibits of coal mining in museums; perhaps the best known is in the Deutsche Museum in Munich. Finally, the city of Beckley, West Virginia, has acquired a small exhausted mine, outfitted it with representative if outdated equipment, and provides tours with lectures on the history and technology of coal mining.

Epidemiology of Coal Mining Populations and Prevalence of CWP

Discussions of coal workers' pneumoconiosis (CWP) without reference to the epidemiology of coal mining populations in general are liable to distort the significance of respiratory disease in this community.

Appalachia is a cultural and historical unity* with geographic boundaries that also map the distribution of coal. The same mountains that enforced isolation when Appalachia was the American frontier in 1825 bore the coal that was mined since the 1830's. The coal, 250 billion tons of it, thereupon became a powerful shaping force upon the region. After the frontier passed beyond Appalachia, the area was geographically isolated from the mainstream of American economic life and trade and there developed an intensely provincial, introspective regional culture. This lifestyle and world view proved inadequate to cope with the sudden push to exploit the region's coal resources in the late 19th century, backed primarily by remote Eastern and British financial backers. The profound social and economic dislocations which followed have persisted to the present day (19,20).

Demographically, these factors of history and geography produced a marked and sustained loss of population from the region, in the largest internal mass movement of population within the United States up to that time. In the 1950's alone, the State of West Virginia lost 417,000 citizens in net migration, and only 251,000 were born to replace them. The entire region lost over a million residents, a fifth of its population, in 1950. Well-established paths of migration led to Baltimore, Pittsburgh, Cleveland, Cincinnati, and ultimately to Detroit and Chicago. Most of the emigrants were young (52% were 18 to 34 years old, compared to 34% of the area population), men (7% excess over women), and more highly educated than those they left behind (but not as highly as those to whom they migrated) (19).

These trends resulted in large part from diminished demand for coal, lack of alternative economic opportunities, and a regional inability to mobilize community resources (20). Automation and the mechanization of coal mining also reduced the work force, from some 800,000 in the 1920's to somewhat more than 112,000 (the present membership of the United Mine Workers of America) (21). In 1974, with the increasing shortage of petroleum available to the United States from foreign suppliers, the price of coal abruptly more than doubled** and Appalachia is once again experiencing an inflow of capital (21). This has meant the entry of many more new young coal miners in the work force, which has resulted in an age distribution skewed to both older middle age and younger early adulthood, with a demographic gap in between (22).

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*Appalachia is comprised of parts of southwestern Pennsylvania, parts of eastern Ohio, all but the northern panhandle of West Virginia, western Maryland, western Virginia, eastern Kentucky, eastern Tennessee, western North Carolina, northern Georgia, and northeastern Alabama. It is slightly larger than England, Scotland, Wales, and Ireland combined and has a population roughly the same as that of Belgium (19).

**To $24 per ton, utility-grade bituminous.

The age-adjusted mortality rates for Appalachia, when compared with the nation as a whole, have shown that heart disease and cancer* have been the leading causes of death in Appalachia, the latter at a significantly lower rate than for the entire United States (23,24). Mortality from pulmonary disease has been increasing relative to the rest of the country, however, as has been infant mortality. The area, as a whole, was relatively late in controlling tuberculosis (23).

Although mining catastrophes, such as the Blacksville No. 1 (West Virginia) disaster of 1972 (6) and, most recently, Black Mountain No. 1 (Kentucky) in 1976, are the most visible in the media, coal mining as an industry has an exceedingly high rate of nonfatal accidental injuries on the job (2,6), and coal miners have a high mortality from accidents (2,25).

For nonaccident causes of death, the data are somewhat confusing. Although coal miners in general have been shown to have a normal life expectancy (26), they have demonstrably high standardized mortality ratios (SMR) in certain causes of death (25). The answer to this paradox is found in the so-called "healthy worker effect." This principle of occupational epidemiology points out that the industrial work force is composed of largely healthy individuals with much less risk of death or serious illness than the population at large (27). Thus the cohort of coal miners, who are under particular selection pressure due to the strenuous demands of their work, should survive well beyond the "normal" life span and should have low SMRs.

To the contrary, miners have an elevated risk for a wide range of diseases, compared to the U.S. male population at large, particularly tuberculosis, cancer in general, lung cancer, respiratory disease in general, gastric ulcer, and syphilis (25). On an intuitive basis, gastric ulcer and syphilis, like the lesser excess of deaths from cirrhosis and the high excess from homicides (25), can be attributed to life-style in a hard-working rural working-class white society (22). The remainder are less easily explained.

The elevated mortality for tuberculosis may reflect the historical prevalence in Appalachia and its delayed control in the area. The British experience does not confirm suspicions of an elevated risk for TB among coal miners (28).

For lung cancer, the situation is somewhat different. There is no reason to suspect than an American coal miner smokes more than his age-matched nonmining male paired counterpart, and he probably smokes less since he cannot indulge his habit underground. Nonetheless, the best data available show an SMR of 192 (meaning a risk factor of 1.92) (25) compared to 107 for his British counterpart (1). This discrepancy is complicated by differences in record-keeping: the British data aggregate several job categories and may have diluted the true figure (29). Thus there is no satisfactory answer to the question of whether coal miners have a truly elevated risk of lung cancer, but there is enough evidence to suspect this in the United States (30).

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*Unusually high concentrations of cancer deaths in Appalachia are few; they include cancer of the cervix, lip, and oropharynx in women, of skin, and bone (24).

The prevalence of coal workers' pneumoconiosis (CWP) in the United States has been a matter of dispute, due to differences in methodology and criteria for radiographic diagnosis. The last, best study in the U.S. was the second "round" of the Interagency Study administered by the National Institute for Occupational Safety and Health. It yielded a prevalence of simple CWP of 10% in a carefully selected representative population of U.S. coal miners (n = 9,000), with 0.4% showing evidence of complicated CWP (progressive massive fibrosis or PMF) (1). The prevalence of simple CWP and of complicated CWP were both highest in anthracite miners (eastern Pennsylvania) and lowest in the Rocky Mountains (14), and higher in face miners than in surface workers. These rates are somewhat less, in general, than those for British and Welsh coal miners (1,14). These figures almost surely reflect exposure spanning at least two decades before the transect.

Autopsy series are clearly biased, because the patient obviously had a terminal condition. Nonetheless, it is of some use to recognize that the prevalence of complicated CWP at necropsy rose with the time spent underground, and that PMF was present in roughly a quarter of West Virginia bituminous miners with CWP who came to autopsy before 1966. Almost all of these miners with PMF had evidence of cor pulmonale. Silicotic nodules were present, almost always as an incidental finding, in 13% of miners with CWP, with or without PMF (30).

Diagnosis, Screening, and Disability Evaluation

The diagnosis of CWP has become more a matter of legal definition than of medical opinion. This is obviously due to the regulations promulgated under the Federal Coal Mine Health and Safety Act of 1969, as amended in 1972 (6,7).

From 1969 to 1972, under the Social Security Administration, which administered "black lung" compensation, the diagnosis of CWP required two criteria: radiologic evidence of pneumoconiosis, and one of three objective indices of functional impairment—radiographic or autopsy evidence of progressive massive fibrosis, objective evidence of ventilatory impairment (by pulmonary function testing), or objective evidence of impaired gas exchange (by arterial blood gas desaturation or reduced diffusion capacity). In 1972 the Act was amended, and its implementation became the responsibility of the Department of Labor. Since then, the requirement for radiographic proof of pneumoconiosis has been dropped and has been replaced by physical examination and a questionnaire on symptoms (based on the similar standardized format of the U.K. Medical Research Council, which emphasizes chronic bronchitis). These changes have been sharply attacked as a regression from objective to increasingly subjective criteria (8).

An apolitical, scientifically derived diagnosis of CWP begins with a working definition, in pathologic terms. Applying and slightly modifying the International Labor Organization's definition of pneumoconiosis to coal provides: coal workers' pneumoconiosis is the accumulation of coal dust, produced by both mining and handling, in the lungs and the tissue reaction to its presence.

Radiology. There is no practical method of detecting coal dust in the lung of the living patient other than by X-ray. Sputum production of coal pigment is compatible with a recent acute exposure and says nothing about cumulative lung burden.

The interpretation of the chest film in CWP relies on the occupational history, since there is no pathognomonic sign for CWP and there is more similarity than difference between the pneumoconioses. It is even not clear why CWP should project a radiographic shadow in the first place, since coal is mostly elemental carbon: a density of 0.4 g/cm^2 is required for coal dust to be barely visible on the chest film, and the total lung burden of dust is demonstrably lower than that required to produce shadows by density of dust alone (9). This strongly suggests that the origin of the "interstitial" pattern of small nodular opacities characteristic of CWP is the local tissue reaction, superimposed on local concentrations of coal mine dust in macules such as those demonstrated for tantalum powder (11,12). There is even some evidence for endogenous iron deposition (13, 14). The standard system for communicating the morphology, nodularity, and severity of radiographic changes in cases of suspected pneumoconiosis is the UICC/Cincinnati* Classification of the Radiographic Appearance of the Pneuoconioses (15b). This necessarily involved system uses a 12-point scale which markedly lowers interobserver variation in interpretation and variation in radiological technique, which have been major problems in pneumoconiosis radiology (1,16). Using this system, it is nonetheless difficult to detect progression of CWP among most miners, even over a decade's time, once the diagnosis is established (1,16).

A characteristic pattern of "pinhead" multiple nodules, usually with smooth edges, is called "simple" CWP. When any opacity is greater than 1 cm in diameter, it is termed "complicated" CWP, or progressive massive fibrosis.

Pulmonary Function. The pathologic picture of CWP, involving bronchiole, arteriole, and alveolar wall, would suggest that simple CWP has a protean effect on ventilation, perfusion, and gas exchange. In point of fact, it usually does not (1,14-17).

In general, coal miners have slightly lower forced vital capacities (FVC) and FEV_1 fractions than do nonminers; these changes do not become statistically significant until the miners have worked underground 30 years or more. The FVC and FEV_1 and lowest, on the average, for anthracite miners and less reduced for bituminous miners, but are not markedly reduced in any large population sample of working miners (1,14-16). The geographic distribution suggests that miners in the far West have essentially no impairment at all (1,8).

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*UICC is l'Union International Contre le Cancer.

Total lung capacity (TLC) and derived residual volume (FVC substracted from a radiographically derived TLC) are both increased among bituminous miners, but the degree of hyperinflation does not correspond well to the UICC/Cincinnati radiographic classification. Where there was evidence of preexisting obstructive airway disease ($FEV_1/FVC < 70\%$), more hyperinflation was present; but this is a natural consequence of obstruction per se. The most reasonable hypothesis explaining this is that a mild traction emphysema results from peribronchiolar fibrosis at the coal macule (1).

Studies of pulmonary mechanics have shown that static compliance is normal in most miners with simple CWP but may be reduced in complicated CWP. On the other hand, dynamic compliance is reduced consistently, and is frequency-dependent in most, implying the presence of small airways disease (1,16,17).

Closing volumes are slightly reduced in nonsmoking miners with simple CWP, compared to nonsmoking controls. Cigarette smoking obliterates any differences in the smoking groups. Thus the degree of small airways disease associated with CWP is overwhelmed by the changes due to smoking (1).

Diffusion capacity is also much reduced by cigarette smoking in all groups, but is within normal limits for nonsmoking miners with simple CWP. This index of gas exchange surface area and permeability is reduced only in complicated CWP (1,17).

Arterial blood oxygen tension in miners with simple CWP is slightly desaturated compared to nonminer controls, but increases to normal with exercise. By contrast, miners with complicated CWP desaturate somewhat with exercise although they may remain nonetheless within entirely normal limits. The alveolar-arterial oxygen gradient, $(A-a)PO_2$, is extremely sensitive to smoking, but is increased compared to smoking-matched nonminers and increases slightly with exercise and correlates this with higher UICC/Cincinnati classification. Even so, the alterations are minimal (1).

Pulmonary hypertension is not a feature of simple CWP, nor of complicated CWP at rest. Pulmonary arterial pressure may increase more with exercise in miners with complicated CWP (1,17), but total pulmonary vascular resistance decreases normally (17). Perfusion radionuclide scanning, on the other hand, is normal in simple CWP but is abnormal uniformly in complicated CWP, with perfusion defects (1).

In summary, simple coal workers' pneumoconiosis is associated with functionally insignificant small airways disease and gas exchange impairment, which are vastly outstripped by the effects of smoking. Complicated coal workers' pneumoconiosis is associated with obstructive airways disease of varying severity, with a restrictive component and alterations in pulmonary perfusion. The functional significance of this impairment is highly variable between patients.

Disability Evaluation. Having radiographic evidence of abnormality describes a condition. Demonstrating functional aberrations describes a disease. Neither contributes an adequate description of the individual's ability to cope with the physical demands placed upon him by his work and recreation (18).

The evaluation of miners for purposes of compensation is based, at present, on impairment—on the demonstrable reduction in respiratory (both ventilatory and oxygenation) capacity—and not truly on disability, which implies an inability to perform at a given level of physical activity. This leads to a quandary: Should asymptomatic miners with evidence of impairment, but not disability in their daily lives, be compensated to the same degree as those who may have less cardiopulmonary impairment but whose lives are more active or whose subjective symptoms happen to have a lower threshold? If not, can a bureaucracy presume to decide what are standard acceptable levels of physical activity or distress? In the absence of a device for measuring psychic anguish, Congress did not settle on the only reasonable course of action, which would have been to link objective impairment with objective evidence that the condition was present and to allow the miner to declare his subjective distress by applying for compensation. Instead, the miner only has to prove impairment—whether it is linked with CWP in fact becomes less important in the eyes of the law than that a coal miner has lung disease, whatever the cause. No comparable legislation exists for sandblasters, chemical workers, hard rock miners, or similar occupations with comparable risks.

For the miner with true progressive massive fibrosis, a significant shortening of life expectancy and a significant incidence of severe respiratory impairment are undisputed. These unfortunate, often older individuals all too often are denied compensation on technicalities or present themselves late in their course, having been too proud or independent to accept disability (7,8).

Smoking certainly complicates the issue of occupational disability. Clearly, smoking will raise the level of morbidity from respiratory disease in any population. Nonetheless, however smoking may contribute to respiratory disease among miners, incidental to or contributory to complicated CWP, there are five considerations to be kept in mind:

- There is no evidence that coal miners as a whole smoke more heavily than other hard-labor workers; they probably smoke less by virtue of the time spent underground.
- Evidence that cigarette smoking is a health hazard has been heavily popularized only since 1964, after many presently

working miners began their habit and were well into their occupational dust exposure.

- The coal mining community is not particularly receptive to medical arguments to quit smoking: their work is unrelieved and heavy, their historical experience with physicians has been unsatisfactory, and the peer-group pressure to smoke (and drink) is high.
- When a near majority of the adult U.S. population smoked for several decades, combined exposure to cigarette smoke and to occupational hazards was the rule, not the exception, and compensation should be treated accordingly; smokers of that era should not be penalized.
- Coal miners are not immune from the same smoking-related respiratory diseases that afflict the population at large; when it is impossible to unequivocally attribute respiratory impairment to an occupational exposure, at least it makes sense to associate the two by proving the presence of CWP before according preferential treatment.

Sources of Variability in Coal Dust Deposition

The four types of coal, in decreasing order of density and economic value, are: anthracite, bituminous, subbituminous, and lignite (which is barely more dense than dried peat). Each type is graded for useful characteristics by "rank."

Coal dust is composed of elemental carbon predominantly, with small quantities of pyrite (FeS_2), magnetite (Fe_2O_4), calcite ($CaCO_3$), dolomite ($CaMg(CO_3)_2$), sulfur, water, and traces of aluminum (30). Quartz is also present in a proportion that varies with the mining operation; more quartz particles are airborne with rock drilling and cutting operations. In general, however, the airborne respirable particles tend to be more carbon and less mineral than the parent seam (presumably more buoyant in air) (30).

The size distribution of respirable particles has been extensively studied in experimental models, and much is known about the aerodynamic behavior of such particles. It has long been known that particles above 10 μm aerodynamic diameter tend to be deposited in larger airways due to inertial impaction. Furthermore, particles of less than 0.1 μm diameter mostly remain airborne due to the Brownian movement phenomenon. Thus, the particles in the mid-range are most likely to penetrate to the alveoli and, once there, to sediment and deposit on the airspace wall (31). The velocity of this sedimentation is expressed by the following equation:

$$\vec{v}_s = \frac{\rho d \vec{\xi}}{18 \mu}$$

where d is the aerodynamic diameter of the particle, $\vec{\xi}$ is the vector of gravitational acceleration, μ is the viscosity of air, and ρ is the density of the particle. Thus the velocity of sedimentation depends not only on particle size but also on density. One may therefore conceive of the distribution of particles from a mixed coal mine aerosol as a sedimentation gradient, with lighter coal particles penetrating deeper and sedimenting more slowly.

Another factor producing separation and heterogenous deposition of a mixed particle dust is the anatomy of the airways, which is a series of bifurcations. Secondary flows and eddies result in local nonuniform flow, slowing of forward velocity, and permit foci of increased deposition at the bifurcation. Thus the behavior of particles in the 0.5 to 2.0 μm range is governed by boundary-layer and laminar-flow phenomena, resulting in disproportionate deposition at bronchial and bronchiolar bifurcations. These phenomena dictate fairly uniform deposition for particles below 1.0 μm but increasing nonuniformity of deposition with increasing inspiratory velocity for larger particles up to 5 μm (32).

Thus the pattern deposition of coal dust in the lungs of any individual miner at any one exposure is a complex function of the geologic characteristics of the coal seam, the coal mining operation being performed, the ventilation of the room where the mining is taking place, and the miner's breathing pattern at the time.

Pathology and Pathophysiology of CWP

The pathophysiology of CWP is closely linked to patterns of deposition of the dust and to mechanisms of clearance.

Simple CWP. The essential, defining pathologic finding in CWP is the coal dust macule, a discrete (occasionally confluent) nonindurated focus of black pigment macroscopically visible under the visceral pleura and on the cut surface of the lung parenchyma.

Microscopically, the coal dust macules are typically in various stages of development. The pigment is mostly intracellular in macrophages, whose persistence has been noted over 10 years after cessation of exposure. The macrophages are concentrated in the peribronchial and periarterial interstitium, where local fibrosis is apparent. Limited dilation of the respiratory bonchioles is common, in a pattern compatible with centrilobular emphysema but without evidence of bronchiolitis. The distribution of macules follows the known patterns of peribronchial and periarterial lymphatic drainage,* and draining nodes typically show heavy accumulations of pigment (14,30,33,34).

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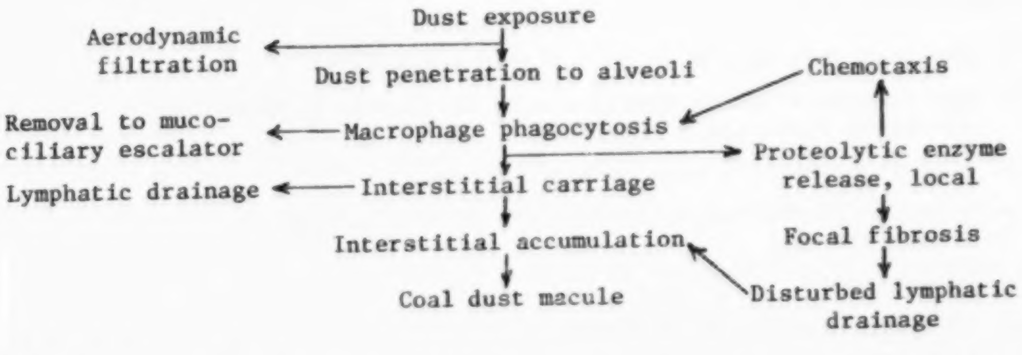
*The lymphatic drainage of the lung consists of local "bronchus-associated lymphoid tissue" draining into "Macklin's sump" at the terminal bronchioles, thence following lymphatics in the pulmonary arterial rays back to the hilum, or following the pulmonary venous lymphatics back to the hilum (35).

Acutely, the dust inhaled is rapidly cleared by the highly effective centripetal mucociliary stream, which removes a prodigious burden. Nonetheless, a quantity is left behind for phagocytosis by alveolar macrophages. The macrophages, having engulfed the individual particles, may migrate on the alveolar surface or interstitially to the edge of the mucociliary escalator (this process is not well understood), or may migrate interstitially. Although interstitial migration probably drains into lymphatics for the most part, a significant amount of dust is trapped in local concentrations at the coal dust macules, where presumably draining is impaired, overwhelmed, or an anatomic "dead end." These transport mechanisms are impaired by inflammatory respiratory disease and by simultaneous exposure to irritant exogenous agents which may be ciliostatic (36), and by exposure to other particulates which may saturate the phagocytic capacity of the macrophage (37).

The pathophysiology of the focal fibrosis and focal emphysema is of some interest, although of limited practical importance because this degree of emphysema in a centrilobular pattern is not clinically significant (33,34). Although one customarily thinks of phagocytosis as a fairly "clean" process, it is not. Phagocytosis of inert particles, such as latex beads, by alveolar macrophages is enough to release small quantities of lysosomal enzymes into the surrounding milieu. These include collagenase, elastase, plasminogen activator, and lysosomal hydroxylases (38). Thus, alveolar macrophages may not be activated by ingesting inert particles such as carbon (39), but they are stimulated to release, inadvertently as it were, potentially locally destructive enzymes (40). Teleologically, this is reasonable in a system evolved to dispatch microorganisms, but it is counterproductive when dealing with inert particles. Furthermore, partially denatured protein is chemotactic, so that the cellular reaction perpetuates itself (40). The schema below is a plausible mechanism for formation and extension of the coal macule.


Dust exposure
Aerodynamic filtration
Dust penetration to alveoli
Chemotaxis
Removal to mucociliary escalator
Macrophage phagocytosis
Proteolytic enzyme release, local
Lymphatic drainage
Interstitial carriage
Focal fibrosis
Interstitial accumulation
Coal dust macule
Disturbed lymphatic drainage

There are certainly opportunities for individual variation implied by this schema (33,36,41). Miners with α_1-antitrypsin deficiency, for example, conceivably might have more extensive local fibrosis than otherwise. Those lacking dynein arms in their tracheobronchial cilia, a defect recently described in Kartagener's syndrome, may have difficulty with mucociliary clearance (42). The avidity and efficiency of macrophages in clearing mycobacteria is a genetic variable in rabbits (43) and may have a genetic counterpart in man.

Complicated CWP, or Progressive Massive Fibrosis. Progressive massive fibrosis (PMF), or complicated CWP, is an occasionally fulminant, usually slowly progressive complication of the advanced grades of simple CWP. In contrast to simple CWP, there is good evidence that PMF shortens life and causes respiratory disability (not just barely detectable impairment (1, 14-16). It is diagnosed radiographically, in which context it is usually referred to as complicated CWP, because PMF generally has been used as a pathological description at autopsy. The criterion is any opacity greater than 1 cm in diameter on X-ray but any nodule greater than 3 cm in diameter at autopsy (41).

Pathologically, PMF arises as a fibrous mass or masses, usually in the upper lobes and posterior segments, heavily pigmented and with a smooth contour. Only rarely do these masses cavitate of calcify, despite their occasionally enormous size; when present, these findings suggest tuberculosis. The nodules are much like gigantic coal dust macules, somewhat more tightly packed. They do not have the whorled pattern so characteristic of silicosis, nor are they stellate as asbestos nodules. Bronchioles are obliterated in the fibrotic mass and vascular structures are invaded by fibroblasts, resulting in an obliterative endarteritis. As the mass expands, larger vessels become involved and in situ thrombosis further compromises pulmonary perfusion (30,34). As the disease progresses, the compliance of the pulmonary hypertension develops, with cor pulmonale and progressive congestive failure (30,41).

The pathophysiology of PMF has been hotly debated; confusion in this difficult areas is made worse by the recognition of at least seven different patterns of fibrosis and simplification in the lungs of coal miners with CWP (30,33,41):

- the coal dust macule (with collagen deposition)
- the silicotic nodule (present most frequently in mine construction crews and roofbolters)
- chronic bronchitis with bronchiolitis
- focal emphysema (often related to chronic bronchitis with bronchiolitis)
- tuberculosis
- rheumatoid (Caplan's) nodules
- histoplasmosis (endemic to much of Appalachia)

Many attempts to associate PMF with either silica or tuberculosis have not proven the point. In both cases, the agents (either silica or acid-fast bacilli) cannot be consistently demonstrated in the PMF lesions or surrounding lung parenchyma, and antituberculosis therapy fails to arrest the process. Although the hypotheses that PMF may result from concomitant exposure to silica or tubercle bacillus are very attractive, based on the known cytotoxicity and fibrosis-inducing behavior of the agents, respectively, the evidence supports neither. It is entirely plausible that infrequent individual cases may be due to mixed CWP and either tuberculosis or silicosis, but these processes cannot be invoked for the majority of cases of PMF on present evidence (30,41). PMF is not related to the total lung or even local burden of dust (1,34).

Other theories of pathophysiology have employed immunologic mechanisms on the model of Caplan's syndrome (to be discussed later). These concepts are still in their germinal stages and await a more complete phenomenological description of the rheumatology of coal miners.

Individual variations in susceptibility to fibrosis have not been well studied for any population, with the exception of α_1-antitrypsin deficiency, which has not been widely investigated in coal miners with PMF. This is a significant omission from the literature and a valid direction for systematic research. A representative sample of miners with simple CWP and PMF should be screened with the following techniques:

- α_1-antitrypsin activity and P_i type
- fibroblast in vitro culture
- hydroxyproline synthesis studies
- history of abnormal fibrosis (e.g., keloid formation, premature wrinkling, family history of such)
- history of exposure to asbestos or oxidizing irritative gases (e.g., NO_2, O_3)
- collagen structure and crystallization (by X-ray diffraction).

These studies should clarify the question of variations in host response, even if the results are negative.

Finally, simultaneous exposure to other agents inducing fibrosis, besides mycobacteria, must be considered. Simultaneous or sequential exposure to nitrogen dioxide (NO_2) with carbon particles works synergistically to modestly increase the severity of centrilobular emphysema which results from NO_2 alone (43), but does not seem to induce nodular fibrosis. NO_2 is a well-recognized mining hazard, generated mostly from welding and shotfiring (4).

The most obvious nonoccupational exposure in miners is cigarette smoking, and there is ample evidence that smoking impairs particulate clearance (44) and predisposes to secondary infection (36,44). Furthermore, the smoking miner is at greater risk for abnormalities in pulmonary function and for chronic bronchitis than his nonsmoking colleague (1,14,15,17). Whether smoking contributes to the development of PMF is problemmatical, but it is certainly a confusing factor which raises morbidity from respiratory disease in any population. There is little reliable published data on the smoking histories of miners with simple CWP compared to those with PMF.

Rheumatologic and Immunologic Aspects of CWP. Caplan's syndrome is the association of CWP, often quite minimal, pulmonary rheumatoid nodules,* and clinical rheumatoid arthritis (RA). The features of the syndrome rarely can be seen in other pneumoconioses. This is the best-defined clinical syndrome and probably represents one end of a spectrum of pneumoconiotic-immunologic disturbances (1,13,44).

Welsh coal miners with PMF have an increased prevalence of RA, but not miners in general. This led to the hypothesis that a population with rheumatoid diathesis may be at increased risk for both PMF and for rheumatoid complications of CWP. The only available markers for such a diathesis are serological, rheumatoid factor (actually several related anti-immunoglobin antibodies) and antinuclear antibody. (These may reflect the early disease state rather than a diathesis, however.) Surveys demonstrated that rheumatoid factor was present in sera of miners with Caplan's syndrome with or without PMF, but not at any unusual frequency in miners with PMF alone, simple CWP alone, or in coal miners at large. On the other hand, antinuclear antibody was present in miners at large, particularly where CWP was prevalent (anthracite much more frequently than bituminous), and at high frequency in miners with either Caplan's syndrome or PMF (1). Thus, the serologic abnormalities appear to indicate the presence of an active immunological process rather than a population at risk.

Antilung antibodies to connective tissue elements have been recognized for some years, but are seen in other chronic lung diseases as well (45). Only Goodpasture's syndrome, where antibody to lung and kidney basement membrane is involved, provides an accepted model for an autoimmune pulmonary disease, and its pathology is quite distinct. It has been proposed that protein adsorption onto coal dust particles may favor induction of rheumatoid factor (46), but the pathophysiological role of this remains obscure.

In short, the immunological aspects of CWP are still at the stage of phenomenological description.

Conclusions

Medical concern over CWP is best directed toward the relatively small fraction of miners with PMF, which is a potentially life-threatening occupational disease. The treatment of advanced PMF is the treatment of cor pulmonale and restrictive lung disease and falls within the competence of any good chest physician. At the present state of the art, the fibrosis cannot be reversed and cannot even be arrested in fulminant cases.* Even removal from the dust exposure has no effect on the progression of the disease.

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*These peripheral, well-circumscribed, occasionally calcified nodules are quite distinctive: they have fibroblasts in palisades, may centrally necrose, and are surrounded by lymphocytes; they may resemble silicotic nodules, and an endarteritis may be present.

The key to decreasing the risk of PMF is to reduce the exposure to all occupational inhalants, including coal mine dust and irritant gases. Reduction of smoking would be desirable, but it is naive to expect this soon. Proposals to employ prophylactic chemical treatments to prevent a pneumoconiosis (47) are questionable to say the least; the experience with aluminum powder as prophylaxis against silicosis is a recent warning that such agents may compound a problem they were designed to correct (48).

Although the incidence of CWP is probably on the decline due to improved dust control, the technology is imperfect and cumbersome. At the moment, the best that can be done is to enforce existing safety regulations, ensure adequate ventilation, limit time spent in more hazardous jobs, and support the technical development of better methods.

A description of the problems of Appalachia appeared recently in the medical literature; like so many, it was oversimplified, distorted, and failed to recognize the substantial progress the region has made since the 1950's. No mention was made of the historically dominant force for medical progress in the region, the United Mine Workers of America,** which is again falling into confusion just as the region as a whole advances. Particularly galling to citizens of Appalachia, and to those who have worked in the region, was the patronizing remark: "...the hillbilly is the most uncomplaining patient in North America" (49); he certainly is not, nor is there any reason why he should be.

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*As knowledge of the pathophysiology of fibroblast injury and proliferation advances, it might be reasonable to design a prospective study as follows: suitable tuberculosis-free patients with fulminant PMF (or silicosis) would be randomly assigned to receive intrabronchial corticosteroids, penicillamine, or aminobenzoate, and followed for arrest of their progression. Such a study should only be conducted by experienced clinical investigators at a major medical center.

**This many-faceted, often difficult institution is responsible for implementing most advances in mine safety and for the indispensable Appalachian Regional Hospital network.

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Appendix L

HOSPITAL RECORD ABSTRACT FORMS

- Short Form (Non-Respiratory & Non-Cardiovascular Diseases)
- Long Form (Respiratory & Cardiovascular Diseases)
- Code List

HOSPITAL RECORD ABSTRACT FORM

Hospital ______________________
Abstractor's Name ______________________
Date Abstracted ______________________

SHORT FORM

(NON-RESPIRATORY & NON-CARDIOVASCULAR DISEASES)

PATIENT'S E.C.I. IDENTIFICATION NUMBER ______________ -1 (1-7)

Personal/Democraphic Data (Write in "unknown" if data unavailable)

1. Patient's Present Residence:
   Town ______________________ ______________ (8-9)
   Zip Code ______________ (10-14)
   Length of Residence (Years) ______________ (15-16)

2. Patient's Previous Residence:
   Town & State ______________________ ______________ (17-18)
   Length of Residence (Years) ______________ (19-20)

3. Date of Birth (Month/Day/Year) ____/____/____ (21-22) (23-24) (25-26)

4. Sex (enter code) M = 1, F =2 ______________ (29)

5. Race (enter code) Cauc = 1, Negro = 2
   Hispanic = 3, Other = 4 ______________ (30)

6. Date of Most Recent Admission (Month/Day/Year) ____/____/____ (8-9) (10-11) (12-13)

   Final Diagnoses: 1) ______________________ H-ICDA Code ______________ (14-17)
   2) ______________________ " ______________ (18-21)
   3) ______________________ " ______________ (22-25)

   Condition at discharge: dead = 1, alive = 2 ______________ (26)
   Family history of the disease or predisposing conditions: Yes = 1, No = 2 ______________ (27)
   If yes, specify ______________________

7. Date of last previous admission (Month/Day/Year) ____/____/____ (29-30) (31-32) (33-34)

   Final Diagnoses: 1) ______________________ H-ICDA Code ______________ (35-38)
   2) ______________________ " ______________ (39-42)
   3) ______________________ " ______________ (43-46)

   Family history of the disease or predisposing conditions: Yes = 1, No = 2
   If yes, specify ______________________ ______________ (47)

8. Date of last previous admission (Month/Day/Year) ______________ (49-50) (51-52) (53-54)

   Final Diagnoses: 1) ______________________ H-ICDA Code ______________ (55-58)
   2) ______________________ ______________ (59-62)
   3) ______________________ ______________ (63-66)

   Family history of the disease or predisposing conditions? Yes = 1, No = 2
   If yes, specify: ______________________ ______________ (67)

HOSPITAL RECORD ABSTRACT FORM

Hospital ______________________
Abstractor's Name ______________________
Date Abstracted ______________________

LONG FORM

(CARDIOVASCULAR AND RESPIRATORY DISEASES)

PATIENT'S E.C.I. IDENTIFICATION NUMBER ______________-1 (1-7)

Personal/Demographic Data (Write in "unknown" if data unavailable)

1. Patient's Present Residence:
   Town ______________________ ______________ (8-9)
   Zip Code ______________ (10-14)
   Length of Residence (Years) ______________ (15-16)

2. Patient's Previous Residence:
   Town & State ______________________ ______________ (17-18)
   Length of Residence (Years) ______________ (19-20)

3. Date of Birth (Month/Day/Year) ____/____/____ (21-22) (23-24) (25-26)

4. Sex (enter code) M = 1, F =2 ______________ (29)

5. Race (enter code) Cauc = 1, Negro = 2
   Hispanic = 3, Other = 4 ______________ (30)

6. Religion (enter code) Prot = 1. Cath = 2
   Jewish = 3, None = 4,
   Other = 5 (specify) ______________ ______________ (31)

7. Marital Status (enter code) Married = 1, Never
   Married = 2, Widowed = 3, Divorced = 4 ______________ (32)

8. Education (enter code) None = 1, Elem = 2.
   Some H.S. = 3, Grad H.S. = 4, Tech/Voc = 5
   Grad College = 7, Grad School = 8 ______________ (33)

9. Insurance Carrier (enter code) UMW = 1, Blue
   Cross/Blue Shield = 2, Medicare/ Medicaid = 3
   Other = 4 (specify) ______________ ______________ (34)

LONG FORM (cont.)

PATIENT NO.________

10. Occupational History

	OCCUPATION (OR DETAILED JOB DES.)	DATES EMPLOYED	NO. YEARS
PRESENT		FROM TO	
PREVIOUS		FROM TO	
PREVIOUS		FROM TO	

Comments:____

History of exposure not mentioned above:________________________

____

____

(36-50)

11. Alcohol Consumption History

	BEER	WINE	HARD LIQUOR
PRESENT CONSUMPTION (Drinks/Day)			
HOW MANY YEARS			
PAST CONSUMPTION (Drinks/Day)			
HOW MANY YEARS			

Comments:____

____

(51-65)

12. Tobacco Use History

	CIGARETTES	CIGARS	PIPE	CHEWING TOBACCO	SNUFF
CURRENT AMT. USED/DAY					
HOW MANY YEARS					
PREVIOUS AMT. USED/DAY					
HOW MANY YEARS					

Brand:________________________

Filter or non-filter______________

____

(66-80)

LONG FORM (cont.)

PATIENT NO.________-2
(1-7)

ADMISSION INFORMATION

Directions: Record all admissions in reverse chronological order. For all admissions involving respiratory or cardiovascular disease, complete a staging form in addition to admission information.

13. Date of Most Recent Admission (Month/Day/Year) ____/____/____
(8-9) (10-11) (12-13)

Final Diagnoses: 1)________________H-ICDA Code________
(14-17)
2)________________ " ________
(18-21)
3)________________ " ________
(22-25)

Condition at discharge: dead = 1, alive = 2 ________
(26)

Family history of the disease or predisposing conditions: Yes = 1, No = 2
If yes, specify________________ ________
(27)

14. Date of last previous admission (Month/Day/Year) ____/____/____
(29-30) (31-32) (33-34)

Final Diagnoses: 1)________________H-ICDA Code________
(35-38)
2)________________ " ________
(39-42)
3)________________ " ________
(43-46)

Family history of the disease or predisposing conditions: Yes = 1, No = 2
If yes, specify________________ ________
(47)

15. Date of last previous admission (Month/Day/Year) ________
(8-9) (10-11) (12-13)

Final Diagnoses: 1)________________H-ICDA Code________
(14-17)
2)________________ ________
(18-21)
3)________________ ________
(22-25)

Family history of the disease or predisposing conditions? Yes = 1, No = 2
If yes, specify:________________ ________
(26)

16. Date of last previous admission (Month/Day/Year) ________
(29-30) (31-32) (33-34)

Final Diagnoses: 1)________________H-ICDA Code________
(35-38)
2)________________ ________
(39-42)
3)________________ ________
(43-46)

Family history of the disease or predisposing conditions? Yes = 1, No = 2
If yes, specify:________________ ________
(47)

17. Date of last previous admisstion (Month/Day/Year)
(49-50) (51-52) (53-54)

Final Diagnoses: 1)________________H-ICDA Code________
(55-58)
2)________________ ________
(59-62)
3)________________ ________
(63-66)

Family history of the disease or predisposing conditions? Yes = 1, No = 2
If yes, specify:________________ ________
(67)

LONG FORM (cont.)

CARDIOVASCULAR DISEASE STAGING FORM

ADMISSION DATE (Mo/Day/Yr) ______/______/______
(35-36) (37-38) (39-40)

CRITERIA	DISEASE ABSENT	DISEASE PRESENT	DISEASE SERIOUS
NYHA Class	I ____ (41)	II ____ (47)	III-V ____ (53)
Clinical	None ____ (42)	Pretibial edema ____ (48)	Angina req. medication, dyspnea on exertion, anasarca, arrhythmias other than PVCs ____ (54)
Diastolic BP	<90 ____ (43)	90-110 ____ (49)	>110 ____ (55)
Chest X-Ray	Normal ____ (44)	CT ratio >1.5 ____ (50)	Pleural effusion, Kerley B lines, vascular congestion, pulmonary edema ____ (56)
EKG	Normal or NSSTTWC's ____ (45)	Any other abnormality ____ (51)	(Per physician's interpretation) ____ (57)
Diagnoses	None ____ (46)	Congestive failure ____ (52)	MI, stroke, TIA, gangrene, claudication, aneurysm ____ (58)

LONG FORM (cont.)

PATIENT NO. ____ (1-7) ____________

ADMISSION DATE (Mo/Day/Yr) ____ / ______ / ____ (8-9) (10-11) (12-13)

RESPIRATORY DISEASE STAGING FORM

For each of the criteria below, check those that apply:

CRITERIA	DISEASE ABSENT	DISEASE PRESENT	DISEASE SERIOUS
Clinical	No mention of coughing, wheezing, dyspnea or hemoptysis ___ (14)	Occasional wheezing and/or coughing ___ (21)	Chronic wheezing, chronic or purulent coughing, chronic shortness of breath, exertion dyspnea, hemoptysis ___ (28)
Chest X-Ray	Normal ___ (15)	Hyperinflation, reduced diaphragmatic excursion, inc. retrosternal space, chronic obstructive airway disease, coal workers' pneumoconiosis ___ (22)	(Per radiologist's assessment) Prog. massive fibrosis, severe coal workers' disease, adv. pneumoconiosis incl. silicosis lung cancer, cavitating lesions honeycombing/severe interstitial fibrosis. ___ (29)
PaO_2 (torr)	>80 ___ (16)	65-85 ___ (23)	<65 ___ (30)
FVC (%Pred)	>60 ___ (17)	40-60 ___ (24)	<40 ___ (31)
FEV_1 (/S)	>2.0 ___ (18)	1.5-2.0 ___ (25)	<1.5 ___ (32)
Diagnosis	None ___ (19)	Diagnosis of emphysema, bronchitis, asthma, bronchopneumonia, acute interstitial pneumonia ___ (26)	Pulmonary edema, bronchiectasis, cor pulmonale, lung abscess lung cancer ___ (33)
Treatment	None ___ (20)	*Aminophylline, Tedral, Marax*, antibiotic prn, IPPB before surgery ___ (27)	Any steroid-prednisone, cortisone, prednisolone, dexamethasone, or other; terbutaline, isoproterenol *(Isuprel)*, cromolyn *(Intal, Aarane)*, mucomist, isoetharine *(Bronkosol)* ___ (34)

CODE LIST

ENVIRO/WYOMING GENERAL PATIENT CODE LIST

ENVIRO	WYOMING GEN.	ENVIRO	WYOMING GEN.	ENVIRO	WYOMING GEN.
1		31		61	
2		32		62	
3		33		63	
4		34		64	
5		35		65	
6		36		66	
7		37		67	
8		38		68	
9		39		69	
10		40		70	
11		41		71	
12		42		72	
13		43		73	
14		44		74	
15		45		75	
16		46		76	
17		47		77	
18		48		78	
19		49		79	
20		50		80	
21		51		81	
22		52		82	
23		53		83	
24		54		84	
25		55		85	
26		56		86	
27		57		87	
28		58		88	
29		59		89	
30		60		90	
91		92		93	
94		95		96	
97		98		99	
100					

Appendix M

EFFECTS OF EXPOSURE TO CARBON DISULFIDE AND HYDROGEN SULFIDE

Table M-1

EFFECTS OF OCCUPATIONAL EXPOSURE TO CARBON DISULFIDE AND HYDROGEN SULFIDE

No. of Workers	Age: Mean or Range	Concentration (mg/cu m)*		Duration: Mean or Range (years)	Effects	Reference
		CS2	H2S			
100	-	450-1,000	-	-	Polyneuritis in 88%, gastric disturbances in 28%	31
43	53	30-1,500	-	21	Encephalopathy	31
107	32	200-400	-	1-9	Ophthalmic pressure 138/110, vs 115/87 in controls	68
185	25-35	62-174	-	ca 5	Eye burning in 96% of rayon-production workers, 44% of cell-fiber workers; pupillary light reactions abnormal	69
100	39	31-137	-	10	Psychomotor and psychologic disturbances	63
125	47	ca 124	-	13	Coronary heart disease in 5.6%, vs 1.2% in controls	48
33	22	40-81	-	2	Asthenospermia, hypospermia, teratospermia	58
350	33	19-65	1-6	6-10	Periodontal changes	10
116	50	>62	-	>5	Coronary heart disease in 16.5%, vs 2.7% in controls	49
28	44	ca 62	-	13	Coronary heart disease in 3.6%, vs 1.2% in controls	48
38	51	29-118**	***	20	Ocular vascular rigidity	66
100	48	29-118**	***	15	Ophthalmic circulation slowed	65

Source: NIOSH, Criteria for a Recommended Standard: Occupational Exposure to Carbon Disulfide, 1977.

Table M-1 (continued)

No. of Workers	Age: Mean or Range	Concentration (mg/cu m)*		Duration: Mean or Range (years)	Effects	Reference
		CS2	H2S			
118	-	29-118**	***	15	Polyneuropathy, abnormal EEG's	56
343	45	29-118**	***	11	Angina in 17%, vs 11% in controls; blood pressure 140/91, vs 136/85 in controls; coronary heart disease cause of 52% of deaths, vs 31.7% nationally	8
343	45	29-118**	***	11	Fasting glucose levels increased with longer exposures; plasma glucose levels higher than in controls	43
319	45	29-118**	***	>10	Coronary heart disease mortality 5.6 times that in controls; total mortality 2.7 times controls	44
322	45	29-118**	***	>10	Coronary heart disease more frequent than in controls: fatal infarctions 4.8, total infarctions 3.7, nonfatal infractions 2.8, angina 2.2, "coronary ECG's" 1.4 times higher than controls	45
343	25-72	29-118**	***	>10	Life expectancy decreased 0.9-2.1 years, depending on age, during 8-year followup	47

Table M-1 (continued)

No. of Workers	Age: Mean or Range	Concentration (mg/cu m)*		Duration: Mean or Range (years)	Effects	Reference
		CS2	H2S			
36	42	29-118**	***	>6	Peripheral nerve and CNS damage; conduction velocities slowed; EMG's abnormal	55
397	35-64	29-118**	***	-	Coronary heart disease cause of 42% of deaths in highly exposed workers, 24% in moderately exposed, 14% nationally	41
165	53	<59**	***	<10	Coronary mortality during 8-year followup 5.8%, vs 2.6% in controls; total mortality 10.2% vs 6.7% in controls	17
630	20-40	31-50	-	-	Immunologic reactions decreased; job absenteeism increased	75
138	<50	22-44	-	<10	Arteriosclerotic changes in 30.4%, hypertension in 23.2%	33
94	18	12-31	<10	<1	Hypotension, nervous system excitability	54
189	<30	ca 28	-	>3	Spontaneous abortions in 14.3%, vs 6.8% in controls; premature births in 8.6%, vs 2.8% in controls	61

Table M-1 (continued)

No. of Workers	Age: Mean or Range	Concentration (mg/cu m)*		Duration: Mean or Range (years)	Effects	Reference
		CS2	H2S			
209	20-40	>22	<10	-	Menstruation irregular, painful, abundant, and prolonged	60
60	25	ca 16	-	-	Muscular power diminished, reflexes slowed	57
500	18-60	<9	-	1-30	Retinal degeneration, conjunctival inflammation, temporary corneal opacities, color-vision disturbances	67
500	20-40	<1	<10	-	Menstruation abundant, painful, prolonged	60
94	18	3-9	<10	<1	Hypotension; nervous system excitability	54

*1 mg/cu m = 0.321 ppm

**These studies are based on the same cohort of workers, exposed to carbon disulfide plus hydrogen sulfide at concentrations averaging 29-88 mg/cu m in the 1960's, 59-118 mg/cu m in the 1950's, and higher before 1950.

***Hydrogen sulfide concentrations are included in those given for carbon disulfide and were estimated to be about 10% of the total.

Table M-2

EFFECTS OF EXPOSURE TO CARBON DISULFIDE OR TO CARBON DISULFIDE PLUS HYDROGEN SULFIDE ON ANIMALS

Route of Exposure	Species	Exposure Concentration*		Exposure Duration	Effects	Reference
		CS2	H2S			
Inhalation	Rat	2,330	0	6 hr/d 5 d/wk 10 wk; then 3 d/wk 12 wk	Lethargy, loss of motor control, slowed MCV's with no recovery in 12 wk	78
"	"	2,330	0	6 hr/d 5 d/wk 2-5 wk	Lethargy, slowed but reversible MCV's	78
"	"	2,000	0	2 hr/d throughout pregnancy	Increased fetal mortality, decreased fertility	81
"	"	1,500	0	5 hr/d 6 d/wk 1-15 mon	Weakness, paralysis, myelin and neuron degeneration, weight loss	79
"	"	12	**	70-110 d before mating and during pregnancy	Increased fetal mortality, terata	88
"	"	1.0 1.0 0.1 0.1	0.1 0 0.1 0	160 d	Inflammation of bronchi, weight changes, increased serum asparatate aminotransferase and blood cholinesterase activities; most severe with combined exposures	85

Source: NIOSH, Criteria for a Recommended Standard: Occupational Exposure to Carbon Disulfide, 1977.

Table M-2 (continued)

Route of Exposure	Species	Exposure Concentration*		Exposure Duration	Effects	Reference
		CS2	H2S			
Inhalation	Mouse	2,000	0	2 hr/d throughout pregnancy	Increased fetal mortality, decreased fertility	81
"	Rabbit	780-2,330	0	6 hr/d 5 d/wk 38 wk	Paralysis, CNS damage, slight liver damage, weight loss	84
"	"	930 930 0	140 0 140	30 min/d 120 d	Abnormalities of bone marrow, kidneys, spleen; decreased spermatogenesis, loss of appetite, blood changes; most severe with combined exposure	86, 87
ip	Rat	78	0	4 mon (every other d)	Testicular lesions, no spermatogenesis	80
"	"	78	0	2 mon (every other d)	Decreased number of spermatozoa; blood vessels engorged, walls thickened	80
"	"	39	0	"	No effects	80

*Concentration given in mg/cu m for inhalation exposures, mg/kg for injections; 1 mg/cu m = 0.321 ppm

**Hydrogen sulfide concentration included in that for carbon disulfide

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